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Table of Contents.

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ORIGINAL ARTICLES— Page.
E. H. Embley Memorial Lecture—Some Current Anæsthetic Problems, by G. R. Troup 841
Studies on Some Clinical Features of Poliomyelitis: I. The Relation of the Clinical Features in Bulbar Poliomyelitis to Tonsillectomy Status, by R. V. Southcott 845
Unidigital Clubbing, with Report of a Case, by Basil A. Stoll, F.F.R., M.R.C.S., D.M.R.T.&D., D.M.R., and W. R. Beetham, M.B., B.S. 852
Myocardial Necrosis and Infarction in Relation to Effort, by D. W. Neville 855
Tennis Heel, by J. Woolnough, M.B., B.S. 857
The Ectopic Ureter, by Douglas Cohen 857
Kernicterus, with Special Reference to Double Exsanguination Transfusions, by Elizabeth K. Turner, M.D. 860

REVIEWS—
Manual of Clinical Mycology 862
History of the Second World War: United Kingdom Medical Series. "The Royal Naval Medical Service" 863
Pomp and Pestilence 863
Spot Diagnosis 864
Medical Mycology 864

BOOKS RECEIVED 864

LEADING ARTICLES—
The Activities of the Cancer Institute of Victoria 865

CURRENT COMMENT—
Hospital Experience and Medical Registration in New South Wales 866
The Effects of Immobilization 866
The Pathogenesis of Acute Pancreatitis 867
Retrolental Fibroplasia 868
Radical Cancer Surgery 869
Peyronie's Disease 869

ABSTRACTS FROM MEDICAL LITERATURE—
Dermatology 870
Urology 871

SPECIAL ARTICLE— Page.
A Children's Clinic in Korea 872

BRITISH MEDICAL ASSOCIATION NEWS—
Meeting of the Federal Council 873
Scientific 873

OUT OF THE PAST 875

SPECIAL CORRESPONDENCE—
New Zealand Letter 876

CORRESPONDENCE—
Ear Complications Associated with Spear Fishing 876
Subcutaneous Injection of Adrenaline during Administration of a General Anæsthetic 876

MEDICAL PRACTICE—
Medical Practitioners Act, 1938-1953 876

OBITUARY—
Frederic Wood Jones 877

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 879

NAVAL, MILITARY AND AIR FORCE—
Appointments 879

POST-GRADUATE WORK—
The Post-Graduate Committee in Medicine in the University of Sydney 880
Seminars at Royal Prince Alfred Hospital 880

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—
New South Wales 880

NOMINATIONS AND ELECTIONS 880

DEATHS 880

DIARY FOR THE MONTH 880

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 880

EDITORIAL NOTICES 880

E. H. Embley Memorial Lecture.[1]

SOME CURRENT ANÆSTHETIC PROBLEMS.

By G. R. TROUP,
Perth.

THE honour of being invited to deliver the E. H. Embley Memorial Lecture has been in the past reserved for one with close academic associations. It falls now to one who lacks such an advantage.

The opportunity of addressing you I owe to the trustees of this memorial lecture, to whom I offer my sincere thanks. When one reads of the life and work of the man to whose memory we pay tribute tonight, so ably related by the first of my illustrious predecessors (Osborne, 1932), the reaction is one of both admiration and humility. Perhaps the best assessment of Embley's worth is in the editorial eulogies in the *British Medical Journal* and *The Lancet* published concurrently with the results of two of his major works. Even when allowance is made for his training by such masters as Martin and Osborne, in a field for which he was initially unequipped, this praise is of particular significance, coming from the source and at the time it did. It is rather ironical that perhaps the greatest appreciation of his research came from outside Australia, and that his example has not been followed within these shores until possibly the last year or so. Figure I, kindly prepared for me by Dr. Geoffrey Kaye, gives some slight but intimate reminder of this remarkable man. It shows two ether inhalers from his own anæsthetic apparatus and some glass equipment that he used in some of his research work at the physiology department of the University of Melbourne. Much of this glass equipment, as Osborne has related, was made by Embley himself. What you see includes practically all the personal links that remain. Appropriately enough, these have found a resting place in the historical section of the museum of the Australian Society of Anæsthetists.

It is my privilege to have lived through a most exciting and enthralling period, in which tremendous changes and, we hope, advances have been made in anæsthesia. By environment and the exigencies of a comparatively new branch of medicine it has been my good fortune to take some active, if minor, part in the initiation of a few of these changes. With such a background, inadequate as it is, and in view of my advancing years, I may be forgiven for choosing to talk about some of the problems that face us, as I see them.

The first of these problems that I wish to discuss is that of teaching. James (1951), that very able teacher and director of an important Australian anæsthetic department, pointed out in the last of these memorial lectures how adequate teaching and practical experience alone can prevent the majority of anæsthetic deaths. Those of us who have sat in judgement on committees inquiring into deaths under anæsthesia appreciate the truth of these words.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on August 4, 1954.

It may seem somewhat odd to choose this occasion to raise the aspect of research in its relation to teaching, but in extenuation it must be remembered that Embley, apart from his research work, was also a teacher. Some criticism has been cast that, in the field of anæsthesia, Australian research has been sadly lacking. This is essentially correct, but it is also true that those few who had the necessary qualifications to be trained in research were busily engaged in trying to render a service to the community, and at the same time meet the demands of undergraduate and post-graduate instruction. Such a state of affairs is likely to remain for some time to come. I submit, at the risk of invoking the wrath of those who think otherwise, that it is not wholly to be deplored. In case by those words I may be accused of insularity, I should like to quote one who must remain anonymous. He comes from overseas and is of international renown. His remarks to me arose from the fact that tremendous advances in anæsthesia over the last two decades had occurred, largely as the result of research. His words were as follows:

> There is, at the moment, much greater need for teaching than research. It strikes me that if we could only disseminate what is already known, there need be no further fatalities in anæsthesia. What tragedies occur happen, not because of want of research, but failure to apply what are well known facts. It is high time for people to keep their feet to the ground.

Those are words on which we all might ponder.

FIGURE I.

Let us view the scene here in our own country. We have a handful of men and women carrying out full-time anæsthesia. They administer the more highly specialized and, in addition, deal with many of the routine anæsthetics. The remainder, a not inconsiderable number, are given by general practitioners.

It has been said that modern anæsthesia is "fatally easy to give", and some truth lies behind that somewhat morbid quip. In the "rag and bottle" era, when drugs were limited and techniques simple, the problem of teaching was comparatively easy. Those days have gone, and drugs are now multiple and techniques often superficially complicated. The repercussions have been devastating and reflect basically on teaching. Modern anæsthesia, when properly applied, apart from making the surgeon's work easier, has, we hope, been of advantage to the patient by reducing anæsthetic mortality and morbidity. For these apparently adequate reasons many surgeons today are unwilling to operate on patients anæsthetized by the old techniques. This factor has depleted the number of cases available for undergraduate teaching to an alarming minimum, for under existing teaching conditions we still have to rely on the "open ether" method for demonstrating to students the signs of anæsthesia. It has reached a degree, in at least one large teaching school, at which undergraduates have to be "farmed out" to other hospitals in which suitable cases are still available for instruction. This creates its own problem in the way of supervision.

The advent of the barbiturates for intravenous administration resulted materially in teaching difficulties. Few today will willingly submit a patient to the "bludgeoning into anæsthesia" by means of a "mask" if he can be safely wafted into unconsciousness with a few cubic centimetres of "Pentothal" introduced into a vein. James has pointed out that this method of induction materially interferes with the signs of anæsthesia, but most of those responsible for undergraduate instruction, including himself, have compromised in making the intravenous administration of a barbiturate the standard teaching for induction of anæsthesia.

I mention these various aspects because I believe that a much greater compromise has to be made if we are to cope with the problems that face us. In the past we have used as a teaching basis the stages and signs of anæsthesia produced by drugs, and admirably laid down by Guedel (1951). The drugs produced the anæsthetic state and the necessary operating condition. The only physiological control was by the quantity used. Today we still use drugs for narcosis and relaxation—modified, however, so that the orthodox signs of anæsthesia no longer exist. The stress is now placed on maintaining as near normal physiological conditions as possible, and various mechanical means are used to attain this end. The change has been a fundamental one. It cannot be said that teaching has altogether kept pace with the change.

That the problem is exercising the minds of those elsewhere is shown in a summing up of the situation which appeared in the *British Medical Journal* early this year (Editorial, 1954). *Inter alia* it comments as follows:

> Difficulty is now being experienced all over the country in obtaining anæsthetists of any sort. There is a shortage of trained men and women in the specialty and how to attract new entrants is at present the concern of the various national bodies dealing with anæsthetic affairs. Many undergraduates are not at present getting the training in anæsthetics they should, in spite of the requirements of the General Medical Council. Some (teachers) indeed, in their frustration, feel that anæsthesia is better kept out of the hands of the non-specialists altogether, and its study limited to post-graduates. But until the number of trained anæsthetists is much greater, the undergraduate must still be taught the subject and taught it well.

Our difficulties here in Australia are even greater. The present number of full-time well-trained anæsthetists is inadequate for metropolitan needs alone. In addition the problem raised by environment in our rural and outback districts probably has no parallel anywhere in the world. Can anything constructive be offered to aid the situation as it exists?

It seems to me that the first essential is to encourage, by all means in our power, the establishment throughout the country of anæsthetic departments in hospitals whose size warrants them. The provision of a director in charge of each, with the necessary qualifications and experience, goes without saying. Australia has lagged behind both Britain and the United States, in which countries the creation of these departments is now accepted as an established necessity. The reason for this has been partly financial, partly scarcity of trained personnel, and partly perhaps an inherent feeling in some quarters that their establishment will encroach on the honorary system and be the thin end of the wedge in the evolution of a bureaucratic system of medicine. I place the departments of anæsthesia first, because without them organization, teaching and research become haphazard and inefficient. I can only speak from intimate experience of the State where I practise; but I cannot imagine that the story differs much elsewhere. The establishment of a department in the main metropolitan hospital in that place changed anæsthetic chaos into some degree of order. A service was provided which had never before been available. I trust I am correct in saying that some who were mildly opposed to the idea now praise the result. The advantage gained by the thirty odd resident medical officers, who for the first time in the history of the hospital have acquired some organized teaching, is very obvious.

The aspect of professorial status for those anæsthetic teaching centres that warrant it may sound ambitious, but it seems at least to be worthy of reflection. Britain has already established several such centres, and the United States many. The acknowledged correlation of anæsthesia with the basic sciences, with medicine and with surgery surely deserves its earnest consideration in Australia.

As to what constitutes an adequate environment for a department, this has as yet to be determined. Decentralization by the creation of the regional or base hospital is one of the practical solutions of hospitalization in our extensive continent. Which of these will warrant a department of anæsthesia, time and experience alone will tell.

FIGURE II.

I have already pointed out the lack of highly trained personnel for executive positions. This brings one to the second essential—namely, the creation of a standard for accepting those willing to enter the ranks of fully trained anæsthetists. The position in Britain and Australia, in this regard, is neither entirely clear nor satisfactory at present. In Britain the creation of a faculty fellowship examination of particularly high requirements, and the retaining of a lower plane diploma, will establish two standards. In Australia we have two diplomas organized by their respective universities. It is possible that the recently created Faculty of Anæsthetists of the Royal Australasian College of Surgeons may make possible entrance to it by examination, when such is feasible. With the present multiplicity and variability of standards in both this country and abroad, one can imagine the uncertainty of the young graduate wishing to take up anæsthesia as his or her life's work. It is doubtless at present preventing some from so doing. It may be that competition, even in standards, is to the ultimate benefit of all. One cannot help feeling, nevertheless, that divided control is to a certain extent a waste of expert manpower, and that history may label it as a mild manifestation of parochialism which should have no place in any branch of medicine.

The creation of what constitutes the specialty of anæsthesia in this country has been largely brought about by the activity of the Australian Society of Anæsthetists. Associated with this group is a unique centre complete with museum, library and ancillary services. It has been established mainly by the altruistic enthusiasm of one man. The utilization of the available facilities there, when combined with the potential activities of the newly established faculty, should provide a vast cooperative source from which much of value in the teaching field should emerge.

"Brown of Adelaide", to whom anæsthesia in Australia owes so much, established many years ago the first committee for inquiring into anæsthetic deaths. Those of us who have followed his example can vouch for the value of this avenue of approach in learning from our mistakes and teaching others how to avoid them. Experience has shown that the confidential nature of the findings allows of full protection, and at the same time ensures that they will not degenerate into mere "white-washing" formalities.

The second problem with which I propose to deal can be summed up in the question: Is simplicity compatible with "modern anæsthesia"? It is with some diffidence that I approach this. A learned professor, well known for his essays, admitted that it was impossible to define simplicity simply. Nosworthy (1953), of our own calling, has traced the difficulties of so doing when applied to anæsthesia. He wisely states that in the final analysis simplicity must be appraised on results. He refers to the current fashion and simplicity of the thiopentone-nitrous oxide-relaxant technique, and points out what pitfalls can occur when this is used by the unwary and relatively untrained. We are all aware of the occasion that he describes as "the minor operation being made the occasion for an unnecessary major anæsthetic". We are all too conscious of "polypharmacy", in which even the skilled and highly trained pharmacologist would find it hard to predict the end result. The "lytic cocktail" may be the latest manifestation of this phase. It is easy to leap to conclusions that history proves to be untenable. I have been particularly struck, on my annual pilgrimage to a highly specialized team in thoracic surgery, to observe that, on each occasion, less and less of fewer and fewer drugs are being used to obtain progressively better end results.

The quips that have been heard over recent years about the "anæsthetic pantechnicons" are not wholly unjustified. Adequate oxygenation has long been recognized as a fundamental essential in anæsthesia; but it is only in recent years that the detrimental effect of inadequate carbon dioxide elimination has been fully appreciated. All anæsthetic drugs cause respiratory depression at some stage,

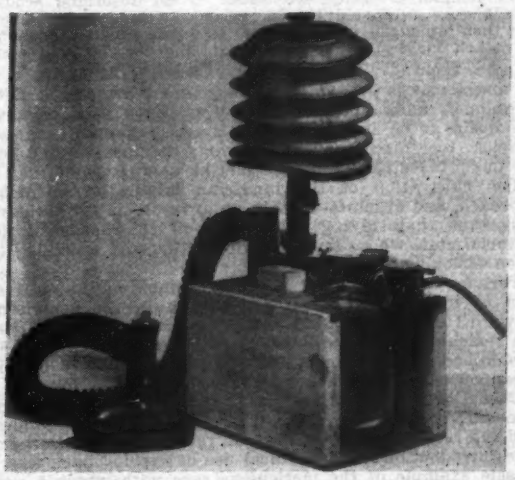

FIGURE III.

and it can perhaps be justifiably laid down that no prolonged anæsthetic, even though it be a simple "ether", should be administered without some means of assisting respiration. No complicated apparatus is required for this purpose. The illustrated Oxford inflating bellows (Figure II) shows how simple such a piece of apparatus can be. Macintosh (1953), who is responsible for it, states that for the last three years he has used only this unit to inflate the lungs with anæsthetic adjuvants, when normal respiration has been suspended, during major thoracic and abdominal surgery. He demonstrated its use in a transthoracic gastrectomy, which some of us saw not far from these walls during his visit to us three years ago. A similar piece of apparatus illustrated in Figure III has been used by me for some years. It was suggested to me by John Watson of this city. Like the Oxford inflator, it

consists of a bellows and two valves ensuring a unidirectional flow. At the inlet is a container to which either ether or "Trilene" can be added. Other modifications have been put forward, notably that of Renton (1952). These illustrations are given merely to indicate the belief that in some aspects of anæsthesia simplicity is not wholly incompatible with modern concepts.

I dwell on this theme of simplicity of apparatus because it seems to me to be intimately bound up with the problem of teaching. I have suggested that in any prolonged anæsthetic, even if it is with "ether", some form of assisted respiration should be in use. If this is so, has not the day arrived when we should discard the "rag and bottle" method in undergraduate teaching, and replace it by instruction with some simple mechanical means, based on a sound physiological foundation? It will at least allow those with the necessary ability to learn a technique which will make them relatively safe in using some of the modern advances. One must reiterate that, until the supply of highly trained anæsthetists is adequate, a large proportion of anæsthetics in this country will continue to be administered by those who have received only the basic training of their undergraduate and hospital resident years.

I pass now to a problem which is not wholly restricted to anæsthesia. The spate of articles appearing in any branch of medicine increases as new journals spring into existence. As Mushin (1948) puts it:

> The struggle of reading and digesting them all is a never ending one. Gradually the eye gets used to picking out the striking headline or important summary for the numbed brain to store.

When one comes to the significance of the written word, one is up against even greater trouble. Moran (1952) is frankly pessimistic when he makes the following statement:

> Human credulity, we reflect, is an absorbing study. It gets entangled in the mind like bind-weed. And then we come out with our own particular branch of black magic. No degree of intelligence, no degree of education is proof against gullibility.

In anæsthesia over the last few years, new drugs, new techniques, and almost new concepts, have been arising. The desire to be first in the field is a very human one, and articles too often appear lacking the background of thorough investigation and scientific appraisement.

The cultivation of the inquiring mind, the scientific approach, and absolute honesty as to result, is surely more important than laying the blame on the heads of an editorial staff whose task of adjudication and assessing is often difficult and at times impossible.

Perhaps one cannot get a better example of the cultivation of these essential attributes than in the person of Edward Henry Embley himself. His refusal to accept either the teaching of Syme (1896) or the findings of the Hyderabad Commission (1902) was equalled only by his incontrovertible arguments against them.

Credulity in matters pertaining to anæsthesia often runs rife with the introduction of new drugs and techniques, and the responsibility is heavy on those who initiate them. The introduction of the clinical use of cyclopropane was a classic example of the meticulous care with which a new advance should be published. Any mistakes that subsequently occurred were in spite of the warnings so ably laid down by Waters and his colleagues of Madison (Waters, Rovenstein and Neff, 1934).

We are perhaps at the moment on the eve of other events in which great care is going to be needed in avoiding the pitfalls of gullibility. It has been said (Wood Jones, 1939) that there are few who in their training towards the sciences are sufficiently well versed in philosophy to ensure that their deductions may be esteemed as properly critical, or even logical, in their presentation. May those few help to guide our steps!

It might be assumed from what has been already stated that the problems of anæsthesia were relatively abstract. On the contrary, the whole field of the subject abounds with the more technical.

One small aspect of a special branch will be selected as an example—namely, that of endobronchial anæsthesia. It is chosen because, by force of circumstance, it is the only avenue in which I have any semblance of excuse for offering comments. Its importance in thoracic surgery lies not only in the aspect of "spill over" and giving the surgeon a quieter operating field, but also in the implications involved by changes in blood chemistry.

From a review of the history of the subject by Mushin and Rendell-Baker (1953), it is somewhat illuminating to realize that probably the first endobronchial catheter, devised about 1871, was adapted from a Tarnier abortion catheter. History does not relate whether it was adapted by a gynæcologist who dabbled in anæsthesia, or by an anæsthetist who had leanings towards gynæcology. It is also interesting to note that Head's endobronchial cannula of 1889 did not materially differ in principle from the Carlen's double lumen catheter of some sixty years later.

Although it is not very relevant, I cannot resist the temptation to relate an occurrence many years ago when, at some considerable pains and laborious expenditure of man hours, I worked out and made a simple, if unphysiological, apparatus for inhalation endotracheal anæsthesia. It became quite popular locally, but had not been in use for long when a colleague, with some justifiable glee, informed me that Flagg years before had published a description of a similar and much more physiological piece. The moral is obvious.

The first mention of "one lung" anæsthesia was made by Gale and Waters in 1931. They utilized a long endotracheal cuffed catheter with a lateral curve at the tip. Its ultimate position in the bronchial tree was more or less "on the blind".

In 1935 the use of bronchial blockers for control of secretions in lobectomies was first described. Magill about the same time introduced his right and left endobronchial catheters with bronchoscope, which allowed direct vision placement; but the catheters were rather restricted in use, because they could be used only in large-sized adults.

Blockage of a bronchus with gauze tamponage, as described by Crafoord, never became very popular.

The latest endobronchial catheter in the field is the Carlen's double lumen catheter, which as its name implies allows of inflation or suction in one or other lung at will. Its efficient use depends on an accurate placement and fit, which in some cases are not easy to attain and keep—or at least, have not been so in my hands.

The advantage of the "blocker" over the endobronchial tube lies in the greater protection from "spill over" in lobe resections by accurate placing of the former. In these days of improved pre-operative preparation of the patient in the "wet" ease and efficient suction during the operation, and in view of the case with which bronchoscopic examination and suction can be carried out at its termination, the advantage of the "blocker" diminishes. Here in Australia "blockers" have never been used very extensively.

Orton, a few years ago, introduced a simple method for blind bronchial intubation. He never published the method, which is to be regretted, but has given me permission to discuss it. Subsequently a series of over 200 cases were reported by Bonica and Hall (1950), who used a similar technique with encouraging results, not only in the orthodox cases of pneumonectomy, broncho-pleural fistula and lung abscess, but also in lobectomies. Orton used the ordinary Magill endotracheal cuffed catheter, but cut a longer bevel on it, so that the upper lobe bronchial orifice, especially on the right side, was not occluded. The size of the tube used depends naturally on that of the bronchus. The cuff is uninflated, because with a tube of the correct size a tight fit of the intubated bronchus results. The orifice of the main stem bronchus on the upper and operated-on lung is also thereby left free for drainage and deflation.

I have used Orton's technique in many cases for what I trust has been the help of the thoracic surgeon with whom it is my privilege to work. A slight modification has been added, in that the bevel is cut on the concavity of the curve of the Magill catheter instead of the side. This saves having to rotate the tube through 90° after the tip is in the bronchus, and the same tube can be used for either the right or the left main stem bronchus. It is remarkable how often the end of the tube will find its proper resting

place, with directional guidance, even on the difficult left side.

The technical production of one-lung anæsthesia has a simple and satisfactory solution in the majority of cases by the use of Orton's method. Unfortunately the problem does not end here. The use of blockers and endobronchial catheters results in some interference with blood chemistry, because of the unproductive circulation or "shunt" in the non-functioning lung or part thereof. This is material while the thoracic cage is intact, and a build-up of carbon dioxide is probably inevitable. Whether adequate elimination of carbon dioxide occurs even after the chest is opened and the lung collapses is open to question. Doubt is strongly aroused in some cases by clinical experience.

I quote this single aspect of a lone branch of anæsthesia, to illustrate an unsolved difficulty. Answers are required for many others covering the whole subject. There is no dearth of opportunity for those who have the will to labour. May their numbers increase and multiply.

Conclusion.

In concluding this survey of some of the problems and difficulties that beset our path, I am very conscious of my shortcomings in view of the high ideals laid down for these memorial lectures in the inaugural address. In any rapidly growing specialty there must of necessity be many problems, and anæsthesia is no exception. The examples that have been chosen are only a few of the many, but on the whole they may be classed as major ones. They have been discussed by one whose only real excuse for such an ambitious project is that he has grown up with the specialty, and has it very much at heart. I humbly ask you to "deem it not all a too presumptuous folly".

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STUDIES ON SOME CLINICAL FEATURES OF POLIOMYELITIS: I. THE RELATION OF THE CLINICAL FEATURES IN BULBAR POLIOMYELITIS TO TONSILLECTOMY STATUS.

By R. V. SOUTHCOTT,
Adelaide.

A LONG RANGE association between bulbar poliomyelitis and previous tonsillectomy was demonstrated by Southcott (1952) and Top (1952). Both of these writers reported that 85% to 90% of patients with bulbar poliomyelitis had previously undergone tonsillectomy. Southcott's (1952, 1953) studies showed that the duration of the association was at least nine to ten years. In the 1947-1948 epidemic of poliomyelitis in South Australia, upon which these studies were based, there were only four non-tonsillectomized patients out of a series of 39 with bulbar poliomyelitis. The writer (1953) mentioned that these four non-tonsillectomized patients had a high incidence of "encephalitic" manifestations, using this term to include both patients showing evidence of cerebral damage and those with oculomotor lesions, as had been accepted in an earlier study of the same epidemic (Southcott and Crosby, 1949). However, that series of non-tonsillectomized patients with bulbar poliomyelitis was too short for far-reaching conclusions to be drawn. A further study was therefore made of the bulbar poliomyelitis cases which occurred in South Australia during the period 1949-1950. There were 30 cases in which the criteria for being classified as bulbar poliomyelitis were fulfilled (Southcott and Crosby, 1949; Southcott, 1953). By conducting an inquiry by postal and other means it has been found that 14 patients had undergone tonsillectomy and five had not, and for 11 patients the tonsillectomy status was not known. This further survey has increased to nine the number of non-tonsillectomized patients with bulbar poliomyelitis available to me for study. It is proposed in the present paper to describe the clinical features of these cases, and to compare certain clinical features between the tonsillectomized and non-tonsillectomized patient with bulbar poliomyelitis.

Various other aspects of the symptomatology of various clinical subgroups of poliomyelitis—"encephalitic", bulbar, spinal and non-paralytic—will be examined in the present paper, in their relationship to tonsillectomy status, and to pathogenesis.

Some writers, notably Baker and his colleagues in the United States of America, use the term "bulbar poliomyelitis" to signify cases of poliomyelitis in which there is any lesion of the brain stem—for example, cases in which there is clinical evidence of an oculomotor involvement. This will be discussed further, later in the text. However, in the present article, as in previous studies (Southcott and Crosby, 1949; Southcott, 1952, 1953) the term "bulbar" will be used to signify lesions confined anatomically to the pons and medulla, as instanced by the clinical manifestations given previously.

THE CLINICAL FEATURES OF BULBAR POLIOMYELITIS IN THE NON-TONSILLECTOMIZED HUMAN SUBJECT.

The writer (1953) mentioned briefly some of the clinical features of bulbar poliomyelitis in the non-tonsillectomized.[1] The clinical histories of the nine non-tonsillectomized patients with bulbar involvement available for study will be given (four from 1947-1948, and five from 1949-1950).

CASE NTBP 1 (CASE 185 OF 1947-1948).—The patient was a female, aged nine years and five months, attending "North Adelaide Girls' School". It is believed that she contracted the infection at school from the patient in Case 105, with an incubation period of thirteen days. The earliest prodromal date was December 7, 1947. She sat next to the patient in Case 105 at his D[2] + 1 until he left at noon. (However, she had also sat next to him at his D - 2, D - 3, D - 4, D - 5, D - 6.) She attended school until 3 p.m. of her D + 1, December 8, 1947. On December 7 she became ill with fever, lassitude and stiff neck. She suffered from these symptoms intermittently until December 13, when she was admitted to the Adelaide Children's Hospital. Pains in the chest had commenced on D + 2 (December 9), and continued up to the time of her admission to hospital.

On December 13, lumbar puncture revealed a normal cerebro-spinal fluid pressure. The fluid was opalescent, with 360 cells per cubic millimetre, 55% being lymphocytes, and 45% neutrophile cells; the protein content was 35 milligrammes per 100 millilitres. She was transferred to Northfield Infectious Diseases Hospital on the same day.

Examination there showed the patient to be a healthy looking girl with a temperature of 102° F.; her pulse rate was 128 per minute, and her respirations numbered 24 per minute. The fauces were clear, the tongue was white-coated. The voice was normal. The neck was rigid. There was

[1] In 1953 the writer stated in error that all four non-tonsillectomized patients with bulbar poliomyelitis in the 1947-1948 South Australian epidemic showed encephalitic or mid-brain signs. Actually three out of the four non-tonsillectomized patients with bulbar paralysis showed encephalitic or mid-brain signs.

[2] The term "D" refers to the onset date (that is, the date of the onset of the earliest prodromata—the earliest prodromal date). This provides a useful method of dating for epidemiological and other work. Thus if "D" is December 7, then D + 2 is December 9, *et cetera*.

limitation of flexion of the lumbar part of the spine. Apart from the neck and spine rigidity, the only abnormality noted in the clinical examination was the possible absence of abdominal reflexes on the left side; the reflexes were definitely present on the right side of the abdomen in both the upper and lower quadrants. No other loss of reflexes was noted, and no limb pareses could be detected.

On December 14 she developed paresis of the right deltoid, biceps and triceps, and also of the left deltoid and biceps. At this stage her legs showed no pareses. The neck was still a little rigid, and painful on flexion.

On December 15 she complained of pain in her left hip, aching in the neck, and headache. This was the first appearance of these symptoms, on D + 8. The voice became nasal. She developed progressive respiratory distress, with rapid shallow respirations, and became very cyanosed. She was placed in the respirator. Considerable improvement of her colour ensued, but she vomited during the day and there was a tendency to inhale vomitus.

On December 16 distension of the abdomen developed; it was considered that some ileus was present. The pulse rate rose to 200 per minute. (This is accepted as indicative of a central circulatory lesion.) Therapy by intravenous drip administration, gastric suction and physostigimine injections was instituted.

On December 17 the abdomen was very distended. The respiratory paralysis was so severe that she could remain out of the respirator for only a few seconds. The pulse rate was between 160 and 200 per minute, the rhythm irregular. On this day, as well as on the previous day, she was irrational or even comatose.

On December 18 the condition of the abdomen was recorded as normal, except that the bladder was full. The third, fifth, seventh and twelfth cranial nerves were intact. The voice had returned, and she was now able to drink without regurgitation of fluid. She was now conscious and cooperative, and her general condition was considerably improved. The pulse rate was about 120 per minute. Fæces were disimpacted from the rectum. The bladder was full, needing catheterization.

On December 19 she was much improved. The pulse rate was mostly about 120 per minute, and had been as low as 100 per minute. Some diaphragmatic movements were noted. No tendon jerks could be elicited.

On December 20 the pulse rate was 110 to 120 per minute. She was able to pass urine. She could take small quantities of fluid by mouth. By December 24 she was able to take semisolids by mouth, and could remain out of the respirator for two minutes. On December 27 she was eating and drinking well, and could remain out of the respirator for four minutes. On December 30 it was recorded that still no movement of her limbs had been observed. On December 31 some slight movement in the upper abdominal wall was noted, thought possibly to be due to commencing diaphragmatic movement.

On January 1, 1948, she appeared well, and was eating and drinking well. However, she remained quadriplegic. On January 2 she developed epileptiform seizures, starting with a rapid movement of the eyelids and a fixed deviation of the eyes to the left and upwards; there were also spasm of the lower jaw musculature, frothing at the mouth, and loss of consciousness. These attacks lasted three or four minutes, after which consciousness returned. It was thought that she was mentally dulled since the onset of these attacks. On January 3 the seizures continued, and became more numerous, being recorded as "almost continuous". Spasms "of the facial musculature particularly on the left side" occurred, and she became unresponsive to stimuli. On January 6 it was recorded "more seizures occurred yesterday but they are not as frequent as they were 2-3 days ago". From January 2 to 6 her temperature was subnormal (95·8° to 96·4° F.). On January 7 her condition was considered as still very poor, the seizures occurring every half an hour or so. The abdomen was slightly distended. The pulse was rapid and of poor volume, and the temperature was still subnormal. On January 8 the pupils were fixed and dilated. Her general condition remained unchanged. Seizures occurred about every hour or so. On January 9 the temperature rose to 101·2° F. in the evening. On January 10 she looked very ill. She had become comatose, and remained pyrexial (temperature 102·4° F., pulse rate about 136 per minute). On January 11 she was still comatose. The fits were less frequent, but the breathing was "mucousy". On January 13 she was comatose and blue. At one stage during the morning the pupils contracted to pinpoints, but then resumed their dilated state. On January 14 it was recorded that œdema of the neck, arms, hands and shoulders had developed, being present for three or four days. The general condition had worsened. The temperature had become subnormal again. The pulse was rapid and weak, and sometimes irregular. When she was taken out of the respirator very slight lower intercostal movement was observed. On January 16 her condition was unchanged. Œdema was still present. She had become incontinent of urine. By January 25 her general condition was worse—"colour very poor at times". Pressure sores had developed on the occiput and buttocks. The urine was acid—"difficult to alkalinize". On this day the pupils were contracted, the left eye being turned outwards and the right eye turned outwards and downwards. "She still responds to stimuli."

On January 29 her condition was the same, apart from slight reduction of the œdema. "Marked squint is present. The pupils are sometimes contracted to pinpoint, sometimes dilated." On January 30 it was recorded that she would talk occasionally, and that the "pupils react slightly". On February 8 it was recorded that the colour was at times very poor; that from time to time she would speak a few words, or cry out occasionally. Her condition continued unchanged. On March 1 her condition was the same, but the temperature was now subnormal. On March 3 her condition was the same. Her colour was poor, and the pulse was weak and rapid. On March 5 her condition was seen to be deteriorating. Her temperature was still below 95° F. The pulse was weak but slow, the pulse rate 40 to 50 per minute. On March 8, 1948 (D + 92), she died.

Autopsy was refused.

Summary.—Earliest prodromal date, December 7, 1947; fatal termination. Encephalitic, bulbar and spinal manifestations. Respiratory and circulatory failure of central (medullary) origin. Quadriplegia. Oculomotor (including supranuclear) and autonomic paralysis. Post-encephalitic degeneration with epilepsy and oculogyric spasms. Bulbar symptoms at D + 8, shown by dysphonia and respiratory paralysis. Circulatory paralysis at D + 9. Death at D + 92. Non-tonsillectomized subject.

CASE NTBP 2 (CASE 565 OF 1947-1948).—The patient was a male, aged twenty years and six months, a farmhand. (This was a rural case.) His earliest prodromal day was January 25, 1948, when he developed "a cold in the head with blocked nose and sore throat". He remained in bed the next day. On January 27 (D + 2) he began to see double and developed some pain in the lower part of his back. There was no headache, vomiting or dysphagia. On January 29 (D + 4) he felt giddy in the morning. He was admitted to Northfield Infectious Diseases Hospital the same day.

Examination there revealed the patient to be a healthy looking young man with a "thick, slurred and nasal voice". There was no neck or spinal rigidity (he had not complained of any neck stiffness). The fauces were normal. The palate moved slightly across to the right on phonation, and reacted briskly to stimuli. There were some nasal obstruction. The mouth was otherwise normal. The temperature was 98·6° F., the pulse rate was 80 per minute, and the respirations numbered 20 per minute. The pupils were large, and reacted to light and accommodation. There was bilateral limitation to the lateral movements of the eyes. The ocular fundi were normal. The heart and lungs were normal. No pareses were detected in his skeletal musculature. The knee jerks and ankle jerks were absent. The triceps jerks were present and equal, but the other arm reflexes were absent. The plantar responses were flexor in type. At lumbar puncture the cerebro-spinal fluid was clear and colourless, containing 10 cells per cubic millimetre, all lymphocytes. The chloride content of the cerebro-spinal fluid was 730 milligrammes per 100 millilitres; the protein content was "very slightly increased".

On January 30 (D + 5) the patient's voice was still nasal, but there was no dysphagia and no pharyngeal pooling of mucus. The pupils were dilated, and did not react to light or accommodation. Right ptosis was present, and some strabismus; diplopia was still present. The triceps and supinator jerks were recorded as present but sluggish; both biceps jerks were absent, and the knee and ankle jerks were absent. No pareses could be detected. He complained of "numbness" of the fingers and toes, but examination showed no alteration of sensation. Fine irregular nystagmus was observed.

At night he complained of slight dysphagia, but there was no pharyngeal pooling of mucus. The pupils remained unreactive, and ptosis was bilateral and pronounced. He tended to fall backwards and to the left. He was considered to be somewhat irrational.

On January 31 his dysphagia was worse. His pupils remained dilated, equal and unreactive. The ptosis was still pronounced, greater on the right. The musculature and reflexes in the limbs were unchanged. His temperature rose to 99·4° F. On February 1 his dysphagia had worsened, but there was still no pharyngeal pooling of mucus. His eyes were recorded as almost fixed—"only slight lateral and

vertical movement" being present. His pupils were dilated, equal and unresponsive to light or accommodation. He was apyrexial. His respiration was normal, and no pareses had developed in the limbs. There was no rigidity of his neck or spine.

On February 2 a weakness of the right sterno-mastoid and triceps was noted. The eyes were fixed; he still complained of diplopia. Some accumulation of saliva had occurred—"relieved by atropine". On February 3 the pareses had extended, now involving both biceps, deltoids and sterno-mastoids. Some thrombophlebitis had developed in the left leg (secondary to therapy by intravenous drip), with rigor and slight pyrexia. It was recorded that past pointing and astereognosis had been "present in the left arm and hand for the last 2-3 days". He continued to cough up copious "mucus and saliva". On February 5 an attempt to pass a Ryle's tube caused an attack of laryngeal stridor and cyanosis. "Phlegm [was] complained of in airway. Tracheotomy was performed; no mucus was seen in the trachea. Respiration satisfactory."

On February 6, at 4 a.m., he suddenly stopped breathing "for no apparent reason". The airway was clear, and no mucus was obtained from the trachea and bronchi on aspiration. Artificial respiration was performed, and his colour returned. His pulse rate was slow, and the pulse was of good volume. He was placed in the respirator. He was irrational and restless all night. The pareses and the astereognosis were unchanged. The thrombophlebitis remained severe. On February 7 he was taken out of the respirator, but became cyanosed, and was returned. Later in the day he became grossly irrational, excited and talkative. On February 8 it was recorded that he was "still making peculiar athetoid movements of the arms. The twitchings of the muscles have diminished since yesterday". The pulse was slow and regular and of good volume. The temperature was 101° F. on that day, and had been 100° to 101° F. over the previous three or four days. "Yesterday there was a large amount of mucus and saliva; less today." The respiration was "normal".

Over the next two days the temperature was 101° to 102·4° F. On February 10 he was less restless, and rational some of the time. The pulse was "satisfactory". The respiration was "irregular but just adequate". On February 12 he was quieter and rational. The pupils remained unreactive, but the movement of the left eye was increasing. The respiration was still irregular, but the amplitude was increased. "Has more control of hands today. Can now swallow a small amount of fluid."

On February 13 he was quieter and rational. The right eye was still immobile, but the lateral and vertical movement of the left eye had increased. The pupils were still dilated, and did not respond to light or accommodation. The swallowing was a little improved. The control of the arms and hands was increasing. The right pupil contracted to pin-point in thirty minutes after the instillation of pilocarpine into the conjunctival sac. On February 14 lumbar puncture was repeated. The cerebro-spinal fluid contained "less than 5 cells per c. mm., all lymphocytes; protein moderately increased". On February 15 some lateral movement was observed in each eye; vertical movement was present only in the case of the left eye. On February 21 the left eye had more medial movement, and the left pupil reacted slightly to light. Good lateral movements were present in the eyes. He was able to swallow well. On February 23 the eyes were the same. His arm movements had improved. The tracheotomy tube was removed. On February 29 both pupils were smaller, the left more so. On March 3 slight lateral movement was observed in the right eye. On March 5 improvements in the power of his neck flexors and arm muscles were recorded. He was swallowing "very well". On March 8 he was apyrexial. He felt very well. His eyes were the same. On March 9 it was noted that his pupils were contracting slightly more, his vision was improving, and he was generally very well.

Subsequently his eye movements continued to improve, his pupils became more reactive, and the ptosis lessened. His muscles improved. He was "walking well" by April 2, and he was discharged from hospital on April 5, though still needing out-patient orthopædic and ophthalmic supervision.

Summary.—Earliest prodromal date, January 25, 1948. Bulbar involvement (palatal paresis) at D + 4. Palatal, pharyngeal and respiratory paralysis. Complete opthalmoplegia (third and sixth cranial nerves, including ptosis). Nystagmus; ponto-cerebellar involvement; paresis of arms and neck flexors. Gradual recovery. Non-tonsillectomized subject.

This patient became irrational before there was any evidence of respiratory paralysis. This early irrationality may be accepted as further evidence of encephalitic involvement. After the development of respiratory paralysis excitability, tremors *et cetera* occurred. The later period of irrationality and excitability may have been secondary to the hypoxia of respiratory paralysis.

CASE NTBP 3 (CASE 690 OF 1947-1948).—The patient was a female, aged forty-five years and four months, a housewife. (This was an urban case.) The earliest prodromal day was March 3, 1948. She was admitted to Northfield Infectious Diseases Hospital on March 6, where the following history was taken: "Has had no food for 4 days. Nausea and vomiting for the last 3 days. Headache and sore throat for 2 days. Voice has been thick for the last 2 days. Difficulty in swallowing for 1 day."

Examination showed the patient to be a healthy looking middle-aged woman. The temperature was 101·6° F. and the pulse rate 90 per minute, and the respirations numbered 16 per minute. There was much redness of the soft palate and oro-pharynx, and much mucopus. There was no palatal palsy. The tongue was furred and moist. The voice was nasal; there was "difficulty in articulation". Eye movements were normal. The pupils were equal and reacted to light and accommodation. There was no lymph glandular enlargement. The heart and lungs were normal. No pareses were detected in the limbs. The reflexes were present and equal, except that the ankle jerks were not elicited. The cranial nerves were intact (except for the voice and throat). There was no neck or spinal rigidity. There was "weakness of the flexors and lateral rotators of the neck". The cerebro-spinal fluid was clear; the pressure was not recorded. Queckenstedt's test gave a normal result. The cerebro-spinal fluid contained 112 leucocytes per cubic millimetre, 90% being mononuclear cells and 10% polymorphonuclear cells; the chloride content was 730 milligrammes per 100 millilitres, and the protein content was not increased.

During the afternoon of March 6 (D + 3), left facial palsy and complete palatal paralysis developed.

By March 7 she had become slightly irrational. Pooling of mucus occurred in the pharynx, relieved by the subcutaneous injection of one one-hundredth of a grain of atropine. Her temperature remained slightly elevated (to 100° F.). Intravenous fluid therapy was continued. There were no apparent pareses in the limbs. By evening the pooling of pharyngeal mucus had increased greatly, and her colour had become poor. Tracheotomy was performed and a large quantity of mucopus was immediately aspirated from the trachea. However, although the respiration was subsequently recorded as "fair", her colour improved but little.

On March 8 (D + 5) her respirations became progressively shallower during the day. She was placed in a respirator in low Trendelenburg position, and continuous suction was maintained from the trachea; the intratracheal administration of oxygen was maintained. Her colour remained poor, and her pulse was of poor volume. On March 9 she was worse. Her colour had not improved, and was now recorded as "cyanosed". The temperature remained elevated. The pulse was irregular and of very poor volume. There was still copious mucopus in the nose and pharynx, but practically none in the trachea. The intravenous drip administration of fluid was satisfactory.

On March 10 her condition was deteriorating. The pharynx and trachea were as on March 9. The pulse was irregular and "of very poor volume", the rate 100 to 110 per minute. She was continuously cyanosed, semiconscious, moving both arms and legs at intervals. On March 12 she was comatose. The pulse was almost imperceptible. For the last few days there had been some blood loss *per vaginam* (menses). On March 13 her condition was generally the same. The loss *per vaginam* had lessened. On March 14 there was some thrombophlebitis in the left leg; the intravenous fluid administration was stopped. The temperature was 104° F. She was somewhat more conscious. The urine was normal. On March 16 she was "mentally bright", and considered to be slightly improved. On March 18 she was "still irrational", and had had a poor night. She was apprehensive and wakeful. The temperature was 99·6° F. Her colour was "fair". She was possibly suffering from early bronchopneumonia, and treatment with sulphadiazine was instituted. On March 20 she was "slightly deteriorated". She was irrational at intervals. The temperature ranged from 100° to 102° F.

On the morning of March 21 she was completely irrational, and her general condition was considered as slightly worse. The temperature was 98° F. The pulse was of "fair volume", the pulse rate 90 to 100 per minute. She was slightly cyanosed. There was "still a large amount of muco-pus coming from the trachea, and a fair quantity from the nasopharynx". The hæmoglobin value of the blood was 11·6 grammes *per centum*; the white cells numbered 9200 per cubic millimetre, 84% being neutrophile cells, 12% monocytes

and 4% lymphocytes. On March 22 there was "general clinical improvement". The X-ray film of the chest, taken with a portable machine, showed "opacities in the lung fields, resembling consolidation, particularly in the subclavicular areas". The ocular fundi were normal. On March 23 her colour was "good". The pulse was "of good volume". The temperature was 100° F. Her condition was considered as "fair". She was able to remain out of the respirator for fifteen minutes. The urine was normal and she was passing it well. She could retain her gastric feeds and sulphadiazine well.

Subsequently she remained irrational and drowsy most of the time. Severe dysphagia remained, with pooling of mucopus. Pressure sores developed over the shoulders and lumbo-sacral area. Penicillin treatment was given from March 6 to 28, and again from April 2 onwards. She died on April 9.

No autopsy was performed.

Summary.—Earliest prodromal date, March 3, 1948. Bulbar symptoms (palatal paresis) first on March 5 (earliest prodromal date to bulbar symptoms, two days). Encephalitic and spinal manifestations also present. Facial, palatal, respiratory and circulatory paralysis; dysphagia. Fatal outcome. Non-tonsillectomized subject.

This was a late metropolitan case, not investigated by field survey.

CASE NTBP 4 (CASE 745 OF 1947-1948).—The patient was a female, a housewife, aged twenty-eight years and nine months. (This was a metropolitan case.) The earliest prodromal date was April 15, 1948. The onset was considered to be of influenzal type. Headache and lassitude were the initial symptoms, the headache lasting three days. At D + 1 some backache occurred, lasting for two days, and becoming severe on D + 3. There was slight nasal discharge on D + 1. There was no sore throat or cough. At D + 2 she became febrile (temperature, 100° to 103° F.), and suffered from nausea and dry retching, but there was no actual vomitus. On D + 3 she had difficulty in speaking; the limbs ached and felt weak. Micturition was normal. She was admitted to Northfield Infectious Diseases Hospital on April 18 (D + 3).

Examination there revealed her to be a healthy looking young woman talking in a very slurred manner. She was not distressed. Her temperature was 103° F., her pulse rate was 128 per minute, and her respirations numbered 32 per minute. The pharynx and fauces appeared normal. There was no pooling of mucus. The soft palate moved "evenly and well" on phonation, and reacted briskly to stimuli. The voice was recorded as "variable—clear at times and then becomes very slurred and difficult to understand". The pupils were equal and reacted to light and accommodation. The eye movements were normal, and no nystagmus was present. The neck and spine were not rigid. The heart, lungs and abdomen were normal. No weakness could be detected in the limbs. All reflexes were equal and active. The plantar responses were flexor in type. Sensation was normal. The cerebro-spinal fluid pressure was normal. Queckenstedt's test produced a normal result. The cerebro-spinal fluid was clear and colourless; it contained 20 cells per cubic millimetre, all being mononuclear; 80% were small lymphocytes and 20% large lymphocytes; the chloride content was 720 milligrammes per 100 millilitres, and the protein content was a little increased; the fluid was sterile.

On April 19 her condition was the same. The temperature was 101° F. and the pulse rate 120 per minute, and the respirations numbered 24 to 30 per minute. Her voice was the same. The palate moved well, and the pharynx was recorded as normal. There had been no pooling of mucus. The limbs, reflexes and eye movements were normal. Apart from the dysphonia, the cranial nerves were intact. On April 20 her general condition was the same. The temperature was settling. She was still very drowsy, but was swallowing well. By April 24 her temperature was normal. Her voice was "gradually improving but she is still very difficult to understand at times".

On May 9 her voice was normal. The limbs and reflexes were normal. She was apyrexial, and she was discharged home.

Summary.—A mild case of purely bulbar poliomyelitis, phonation only being affected, without encephalitic or spinal manifestations. Interval between earliest prodromal date and bulbar involvement, three days.

This was a metropolitan case, and the patient had numerous human contacts in her suburb. It was not investigated by field survey.

CASE NTBP 5 (B.P. 7 OF 1949-1950).—The patient was a soldier, aged twenty-seven years and seven months. (This was a metropolitan case.) The earliest prodromal date was August 25, 1949. He was admitted to the Repatriation General Hospital, Springbank, South Australia, on August 31, where the following history was taken. Six days earlier he had felt tired. He had spent the previous two days rather strenuously. Prior to that he had been well. A severe low backache across the sacrum had developed forty-eight hours prior to his admission to hospital, also headache and general malaise. He had been unable to pass urine since 5 p.m. on the previous day (that is, for nineteen hours). Three hours prior to his admission to hospital he had noticed difficulty in walking. His backache was becoming more severe. He was admitted to hospital as a "stretcher case". On examination of the patient his temperature was 101·6° F. He looked "toxic and flushed". The pupils were equal and reacted to light and accommodation. Apart from a dry tongue, no abnormality was detected in his mouth and throat. No lymph glands were palpable in the neck. The heart and lungs were normal. The pulse was full and bounding. The blood pressure was 140 millimetres of mercury, systolic, and 90 millimetres, diastolic. The bladder was distended almost to the umbilicus; no other mass or viscus was palpable in the abdomen. Examination of the central nervous system revealed partial lower motor neuron paresis with weakness of the following groups of muscles: (i) the extensors of the elbows, the left being more affected than the right; (ii) both quadriceps; (iii) the extensors of the ankle joints, the right being more affected than the left. The triceps and biceps reflexes were absent. The radial reflex was absent on the right, present on the left. The quadriceps reflexes were absent. The Achilles reflexes were present but weak. Of the superficial reflexes, the abdominal and cremasteric were absent; the plantar reflexes were present and flexor. Moderate neck rigidity was present. At lumbar puncture the cerebro-spinal fluid pressure was 240 millimetres. Queckenstedt's test produced a normal result. The fluid was opalescent, and contained 680 leucocytes per cubic millimetre, lymphocytes predominating; its protein content was 150 milligrammes per 100 millilitres.

A diagnosis of acute anterior poliomyelitis was made. Slow bladder decompression by an in-dwelling catheter was commenced, and treatment by sulphacetamide was instituted. He was transferred to the Northfield Wards of the Royal Adelaide Hospital (formerly the Northfield Infectious Diseases Hospital), where treatment was continued.

By 8.10 p.m. that day his respiratory rate had risen to 48 per minute, the respirations being shallow. No diaphragmatic movement could be detected. Very slight thoracic excursion was present. He was placed in the respirator, and supportive treatment was given. On September 1 his general condition was unchanged. Much mucus was present in the throat, and he was unable to swallow. There was difficulty in feeding him by intragastric tube, the fluid returning immediately. Cyanosis was observed periodically.

On September 2 his temperature was 104·2° F. "Suppression of the urine" had occurred. Much mucus was present in his pharynx, "causing frequent attacks of cyanosis relieved by nasal oxygen". He died on the morning of September 2. There is no record of any autopsy.

Summary.—Clinically, a severe bulbar case with involvement of the *nucleus ambiguus* and respiratory centre. Pulse rate never above 128 per minute, except terminally, when it rose to 142 per minute. No evidence of any ophthalmoplegia. Spinal involvement. Inquiry by circular questionnaire in 1952 revealed that he had not been tonsillectomized.

CASE NTBP 6 (BP 9 OF 1949-1950).—The patient was a female child, aged five years. (This was a metropolitan case.) The earliest prodromal date was September 2, 1949. She was admitted to the Northfield Wards of the Royal Adelaide Hospital on September 8, 1949, where her mother gave the following history:

> Six days ago she became listless with a sore throat and slight headache. This improved but the sore throat recurred 3½ days ago and she was treated by sulphadiazine. Yesterday she was drowsy. Today she is much better, but she now complains of pain in the left arm and cannot move it. She has been off her food, but is better now and ate well today. She has walked today. Her general health has been good.

On examination, the patient was cooperative. Her throat appeared slightly reddened. She was able to swallow. Her pupils were equal and reacted to light. No abnormality was detected in the ears. The heart, lungs and abdomen were normal. The knee jerks and ankle jerks were normal. The plantar responses were flexor. She was able to sit up. Kernig's sign was doubtfully present. The cerebro-spinal fluid pressure was "markedly raised" at lumbar puncture. The fluid contained 15 to 25 leucocytes per cubic millimetre, the cell types not being specified; the protein content was not increased.

On September 9 she was "fairly miserable", and the examiner recorded a "definite weakness of the left arm". On September 13 she was comfortable, and the next day she was using the left arm. On September 15 "power was present in the left hand", but she had "lost deltoid, biceps and triceps in the left arm". Weakness was doubtfully present in the external rectus muscle of the right eye, and in the facial muscles. She was able to sit up.

On September 25 it was recorded that the squint was unchanged, but the condition of the left arm was improving. She was transferred to the Adelaide Children's Hospital on September 29. There examination showed her to be "a plumpish happy little girl". The pupils were equal and active. There was paresis of the right sixth cranial nerve. The fauces were red, the tonsils were a little enlarged, the ear drums were normal, the buccal muscles were normal, the teeth were good and the tongue was coated with some white fur. There was no neck rigidity. The tonsillar lymph glands were palpable in the neck. The heart, lungs and abdomen were normal. All gross movements of the left arm were weaker than those of the right. The right biceps jerk was normal, the left was not obtainable. The legs were normal, the knee and ankle jerks were normal, and the plantar responses were flexor in type.

Summary.—Bulbar poliomyelitis with involvement of the right sixth and seventh cranial nerves, without dysphagia. Left arm palsy also present. Earliest prodromal date, September 2, 1949. Bulbar onset, September 15; earliest prodromal date to bulbar symptoms, thirteen days. Circular questionnaire revealed that the patient had not been tonsillectomized.

CASE NTBP 7 (BP 11 OF 1949-1950).—The patient was a schoolboy, aged five years and eleven months. (This was a metropolitan case.) The earliest prodromal date was September 10, 1949. He was admitted to Northfield Wards, Royal Adelaide Hospital, on September 17, 1949, with the following history. He had been well until a week earlier, when anorexia and slight pyrexia were noted. He was well again the following day, and returned to school on September 14, quite well. On the morning of September 15 he had had some fever and sore throat. He was examined by his doctor, and tonsillitis was diagnosed. Treatment with sulphonamide tablets was instituted. His condition remained much the same until 4 p.m. on September 17, when he developed some ocular palsy, and his eye was "sunken". His colour became "nasty" and he had difficulty in swallowing. He had been vomiting for two days. He had had a pain in the back of the neck for two days, but none in the back or limbs. His bowels had not been opened for three days.

Examination of the patient revealed him to be a young boy, of flushed appearance, in respiratory difficulty. There was "pharyngeal paresis—much mucus in the back of the throat". Examination of his eyes revealed "?ocular palsy", but no more precise comment on this is recorded. The neck was very stiff. The physical signs in the lungs were considered as indicating "left lower lobar consolidation". No comment was made on the voice. Cerebro-spinal fluid examination showed that it contained 25 leucocytes per cubic millimetre, 50% being polymorphonuclear cells and 50% lymphocytes; the protein content was not increased. Treatment by penicillin, soluble sulphathiazole and the intravenous administration of fluids was instituted.

On September 20 he was "still bubbly in pharynx. Vomited today and inhaled a piece of vomitus, which caused cyanosis and dyspnœa. Fed by gavage tube". By September 24 he was able to take fluids and solids by mouth. On September 28 his swallowing was much improved, but he was "still protruding jaw and tongue". On October 1 the physiotherapist reported considerable weakness of both sternomastoids, neck flexors and abdominal muscles, and also of the left deltoid, the right rhomboid and the right brachialis to a less extent; the back muscles were also weak.

On October 4 he was transferred to the Adelaide Children's Hospital. Examination there revealed that his speech was slow and rather nasal in tone. Weakness was recorded for various muscles. No comment was made on his eyes. His convalescence was interrupted by rubella, chicken-pox, mumps, acute bronchitis and a bowel infection, in succession. There was a family history of tuberculosis. He was discharged from hospital on June 28, 1950.

Summary.—Earliest prodromal date, September 10, 1949. Bulbar involvement (dysphagia) on September 17; interval seven days. Respiratory paralysis probably present (respirations as high as 56 per minute on September 17). Some ophthalmoplegia undoubtedly present; but insufficient detail in case notes to clarify its nature. Spinal involvement; whether encephalitic involvement was present is doubtful. Non-tonsillectomized subject (circular questionnaire survey).

CASE NTBP 8 (BP 24 OF 1949-1950).—The patient was a housewife, aged twenty-nine years and nine months. (This was a rural case.) The earliest prodromal date was on December 14, 1949. She was admitted to the Northfield Wards of the Royal Adelaide Hospital on December 19, 1949, where the following history was taken. Five days earlier she had become "grizzly" and had had difficulty with vision. The next day she felt nauseated, and vomited. She had vomited several times since. A severe occipital headache developed, which lasted two days or so. The day before her admission to hospital she developed a burning pain in both arms. Her arms now felt heavy. She still had trouble in focusing. She had had a pain in the neck for two days. Her appetite was poor, and her bowels had been constipated. There had been no dysuria. She had been drowsy. All the other members of the household had been well, including her three children.

On examination, the patient was not distressed, and was lying quietly in bed. She was "unable to move the eyes to the left of the midline—the left external rectus muscle being particularly affected". The palate moved evenly on phonation. The fauces were normal. The tongue had a central white fur. The skin was "dry with a slight yellow tinge". The heart and lungs were normal; the blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. The diaphragmatic movements were normal, and the abdominal musculature was strong. There was no obvious limb paresis. She was able to sit up unaided. The reflexes were normal, the plantar responses being flexor in type. Her back and neck were stiff. Pain was complained of in the lumbar region on flexion of her back, and in the shoulders also. Kernig's sign was present. Lumbar puncture revealed blood-stained cerebro-spinal fluid at normal pressure. The fluid contained numerous erythrocytes and only occasional leucocytes.

On December 21 she was "settling down". On December 22 she was afebrile. On December 23 it was recorded that she "still has paresis of external recti" (there is a discrepancy here from the earlier description). On December 26, "Much the same. Eyes I.S.Q.". On December 31, "Eyes I.S.Q. Has weakness of legs". On January 4, 1950, "Patient well today although she still has stiffness of legs and a little blurring of vision". On January 8 she was transferred to the Royal Adelaide Hospital.

Summary.—Earliest prodromal date, December 14, 1949. Bulbar and spinal poliomyelitis. In spite of some discrepancy in the account of the ophthalmoplegia, sixth cranial nerve certainly involved. In addition, blurring of vision and difficulty in focusing indicate oculomotor lesion. No dysphagia recorded. Earliest prodromal date to bulbar paresis (abducens lesion), 0 days—that is, initial ophthalmoplegia. Subject not tonsillectomized (circular questionnaire).

CASE NTBP 9 (BP 28 OF 1949-1950).—The patient was a male infant, aged three years and ten months. (This was a metropolitan case.) The earliest prodromal date was January 8, 1950. He was admitted to the Northfield Wards of the Royal Adelaide Hospital on January 10, 1950, with the following history: "For the last 2 days he has been drowsy and feverish. No other symptoms, but vomited for the first time on admission." The report on the examination of the patient was as follows:

> The patient was too sick to be fully examined. Temperature 103° F., pulse rate 144 per minute, respiratory rate 52 per minute. His throat was sucked out and much vomitus was obtained from the trachea; he was given oxygen by mask. His eyes showed a gross nystagmus. Diaphragms and intercostals functioning.

He was admitted to hospital about 8 a.m. Tracheotomy was performed at 12 noon, and after the insertion of the tracheotomy tube his condition was "much improved". He died at 2 a.m. on January 11. There is no record of any autopsy.

Summary.—Earliest prodromal date, January 8, 1950. Bulbar poliomyelitis with nystagmus, and respiratory and probably circulatory failure. Subject not tonsillectomized (circular questionnaire).

INCIDENCE OF OPHTHALMOPLEGIAS IN BULBAR POLIOMYELITIS.

The ophthalmoplegias seen in the course of routine clinical examination in poliomyelitis may be listed as follows: (i) oculomotor lesions, as shown by any one or more of the following (a) paresis of superior, inferior or medial rectus, (b) ptosis, (c) paresis of accommodation, as shown by difficulty in focusing, (d) pupillary paralysis, as shown by unilateral or bilateral pupillary dilatation, together with interference with the pupillary light reflex; (ii) abducens lesions, as shown by paresis or paralysis of the lateral rectus muscle.

Although trochlear lesions occur, they are difficult to evaluate in a severely ill patient, particularly in the presence of an oculomotor lesion (see also Matske and Baker, 1952).

Although supranuclear lesions occur, the great majority of the lesions are of the nucleus concerned. As has been stated earlier, some writers, notably Baker and his colleagues, accept an oculomotor lesion as a criterion of bulbar involvement in poliomyelitis. In the present paper such a criterion is not accepted, and the figures given in subsequent tables and calculations depend on criteria stated earlier (Southcott, 1953). Using these criteria, we may compare the incidences of oculomotor and abducens lesions in bulbar poliomyelitis, divided between tonsillectomized and non-tonsillectomized subjects over the period from 1947 to 1950. South Australian figures are given in Table I.

TABLE I.

The Incidence of Ophthalmoplegias from Oculomotor and Abducens Lesions in Tonsillectomized and Non-Tonsillectomized Subjects of Bulbar Poliomyelitis for the Periods 1947–1948 and 1949–1950 in South Australia.

Years.	Tonsillectomy Status.	Oculomotor Lesions.	Abducens Lesions.	Total.
1947–1948	Tonsillectomized	4/35 (? 6)[1]	2/35 (? 5)	6/35 (? 11)
	Non-tonsillectomized	2/4	1/4	3/4
	Total	6/39 (? 8)	3/39 (? 6)	9/39[2] (? 14)
1949–1950	Tonsillectomized	1/14	0/14	1/14
	Non-tonsillectomized	1/5 (? 2)	2/5 (? 3)	3/5 (? 5)
	Not known	1/11	1/11	2/11
	Total	3/30 (? 4)	3/30 (? 4)	6/30 (? 8)
Total	Tonsillectomized	5/49 (? 7)	2/49 (? 5)	7/49 (? 12)
	Non-tonsillectomized	3/9 (? 4)	3/9 (? 4)	7[3]/9 (? 8)
	Not known	1/11	1/11	2/11
	Total	9/69 (? 12)	6/69 (? 10)	15/69 (? 22)

[1] The figures in parentheses refer to the possible maximum incidence of that lesion, as sometimes it was doubtful whether such a lesion was present; thus there may have been six tonsillectomized subjects in 1947–1948 with an oculomotor lesion *et cetera*.

[2] The total bulbar cases for 1947–1948 is given as 39, and not 40, as the details of one case were insufficient for this type of symptom analysis, and the tonsillectomy status was unknown: this case is omitted.

[3] The total here is shown as 7/9 and not 6/9, as in one case (NTBP 7) there was some ophthalmoplegia, but the nature of the lesion is uncertain.

To test the significance of the differences in the proportions of these ophthalmoplegias between the tonsillectomized and non-tonsillectomized subjects we proceed as follows. By inspection the proportions observed for 1947-1948 do not differ significantly from those of 1949-1950. We may therefore pool these figures and test them as shown in Tables II, III and IV (ignoring the "not known" class).

Performing an exact test on the figures in Table II, we find that $P = 0{\cdot}014$*.

Performing an exact test on the figures in Table III, we find that $P = 0{\cdot}07$, which is not significant.

Performing an exact test on the figures in Table IV, we find that $P = 0{\cdot}0166$*.

We have thus found, ignoring the "not known" class, that there is a significantly higher proportion of ophthalmoplegias in the non-tonsillectomized than in the tonsillectomized bulbar paralysis subjects. This is so for the abducens lesions. For the oculomotor lesions we may regard the difference in the proportions as suggestive ($P = 0{\cdot}07$), but not quite significant at the 0·05 level of probability.

There appears no reason to believe that the proportion of tonsillectomized subjects should differ in those not returning the postal circular *questionnaires*. At the time when the survey was conducted, no information was available that prior (long interval) tonsillectomy could exert any influence, and other questions were included so as not to draw too much attention to tonsillectomy. It was shown earlier (Southcott, 1953) that in the case of the 1947-1948 figures the proportions of tonsillectomized subjects did not differ between those investigated by early and subsequent surveys.

CLINICAL EVIDENCE OF OTHER CRANIAL NUCLEAR LESIONS AND THE RELATIONSHIP TO TONSILLECTOMY.

Various other cranial nuclei may be involved in bulbar poliomyelitis. It is proposed in this section to examine the incidence of these lesions for South Australian cases over the period 1947 to 1950, in relation to tonsillectomy status. With the fourth cranial nerve nucleus I have insufficient data for such a study. The observed incidence of paresis in muscles supplied by the trigeminal nerve is also low, and in any case the proportions found must be very subjective, depending largely on what attention is devoted to detailed muscle testing, and hence likely to vary considerably between different observers. The figures I have available are small, and insufficient for testing against tonsillectomy status. Brown, Baker, Adams and McQuarrie (1947) also report a low incidence of fifth nerve palsies in bulbar cases. The clinical features of these are discussed by these authors, and by Baker (1949*b*). Lesions of the seventh cranial nucleus are common, and the figures available are sufficient for testing against tonsillectomy status. Lesions of the *nucleus ambiguus* are the commonest nuclear lesion in bulbar poliomyelitis, and the figures will be tested. Hypoglossal involvement is uncommon, and the figures available to me are insufficient for testing against tonsillectomy status; the clinical assessment of such lesions also is somewhat subjective. The assessment of

TABLE II.

All Cases of Ophthalmoplegia.

Tonsillectomy Status.	Ophthalmoplegia. Present.	Ophthalmoplegia. Absent.	Totals.
Tonsillectomized	7	42	49
Non-tonsillectomized	5[1]	4	9
Total	12	46	58

[1] Includes two patients, each with both an oculomotor and an abducens lesion.

TABLE III.

Oculomotor Lesions.

Tonsillectomy Status.	Oculomotor Lesion. Present.	Oculomotor Lesion. Absent.	Total.
Tonsillectomized	5	44	49
Non-tonsillectomized	3	5	8[1]
Total	8	49	57

[1] Omits Case NTBP 7, since oculomotor lesion uncertain.

TABLE IV.

Abducens Lesions.

Tonsillectomy Status.	Abducens Lesion. Present.	Abducens Lesion. Absent.	Total.
Tonsillectomized	2	47	49
Non-tonsillectomized	3	5	8[1]
Total	5	52	57

[1] Omits Case NTBP 7, as abducens lesion uncertain.

central respiratory and circulatory paralysis is somewhat subjective, and the figures will not be tested here. Later in this study evidence will be presented that in bulbar poliomyelitis the medullary vomiting centre is involved. Those cranial nuclear lesions will be presented for which the figures available on incidence are considered suitable for statistical testing for homogeneity between the tonsillectomized and non-tonsillectomized classes.

Lesions Involving the Seventh Cranial Nucleus.

The figures for the observed incidence of lesions of the seventh nucleus, as shown in the facial musculature, may be considered as comparatively objective. The figures for the incidence of such lesions in bulbar cases for the periods 1947-1948 and 1949-1950 in South Australia, divided by tonsillectomy status, are shown in Table V.

TABLE V.

Incidence of Lesions Involving Facial Nucleus in Bulbar Poliomyelitis, for 1947–1948 and 1949–1950 in South Australia, Divided Between Tonsillectomized and Non-Tonsillectomized Subjects.

Years.	Tonsillectomy Status.	Facial Lesion.		Total.
		Present.	Absent.	
1947–1948	Tonsillectomized	11 (? 12)	24 (? 23)	35
	Non-tonsillectomized ..	1	3	4
	Total ..	12 (? 13)	27 (? 26)	39
1949–1950	Tonsillectomized	7	7	14
	Non-tonsillectomized ..	0	5	5
	Not known ..	2	9	11
	Total ..	9	21	30
Grand total	.. .. ..	21 (? 22)	48 (? 47)	69

Testing the figures for the classes shown in Table V in a series of exact tests for homogeneity of proportions of 2 × 2 contingency tables, between 1947-1948, 1949-1950, facial lesion present, facial lesion absent, tonsillectomized, and non-tonsillectomized, we do not find any significant difference (at the 0·05 level of probability) between these distributions. Thus we have not been able to demonstrate, from the admittedly small figures available, any association between tonsillectomy status and the contraction of a facial lesion in bulbar poliomyelitis.

Vestibular Lesions.

Lesions of the vestibular part of the eighth cranial nucleus and its connexions are difficult to test in established cases of poliomyelitis. However, if we accept that a record of nystagmus indicates a vestibular lesion, we may test the proportions observed for homogeneity between tonsillectomized and non-tonsillectomized bulbar subjects. The figures available are subjective, depending on the degree to which nystagmus is looked for, and on the assessment of the observation. Table VI gives the incidence of nystagmus observed in bulbar poliomyelitis cases, divided between tonsillectomized and non-tonsillectomized subjects, between 1947-1948 and 1949-1950 in South Australia, the latter period being divided again into the first and second halves of 1949-1950. As is indicated in Table VI, the incidence of nystagmus in bulbar poliomyelitis is low. Brown, Baker, Adams and McQuarrie (1947) similarly comment on their extensive series, "nystagmus was observed in several patients".

An examination of the figures in Table VI, the "not known" class being ignored, fails to show any evidence of an association between nystagmus incidence and tonsillectomy status. The figures available are too small to show anything except an extremely large difference, if it were present.

Examination of the figures in Table VI reveals one interesting feature. There is no evidence in these figures

TABLE VI.

Incidence of Nystagmus Observed in Bulbar Poliomyelitis for Tonsillectomized and Non-Tonsillectomized Classes, for 1947–1948 and 1949–1950 in South Australia.

Year.	Tonsillectomy Status.	Nystagmus.		Total.
		Present.	Absent.	
1947–1948	Tonsillectomized	3 (? 4)	32 (? 31)	35
	Non-tonsillectomized ..	1	3	4
	Total ..	4 (? 5)	35 (? 34)	39
1949–1950	Tonsillectomized	1st half 0, 2nd half 5 → 5	1st half 5, 2nd half 4 → 9	14
	Non-tonsillectomized	1st half 0, 2nd half 1 → 1	1st half 3, 2nd half 1 → 4	5
	Not known ..	1st half 1, 2nd half 3 → 4	1st half 6, 2nd half 1 → 7	11
	Total ..	1st half 1, 2nd half 9 → 10	1st half 14, 2nd half 6 → 20	30
Grand total	.. .. ..	14 (? 15)	55 (? 54)	69

of change in the incidence of nystagmus until the second half of the 1949-1950 bulbar series. Testing the proportions shown for homogeneity of the proportions tonsillectomized between the first and second halves of the 1949-1950 bulbar series, we have the figures shown in Table VII.

TABLE VII.

Half Series.	Nystagmus.		Total.
	Present.	Absent.	
First half of 1949–1950	1	14	15
Second half of 1949–1950 ..	9	6	15
Total	10	20	30

With an exact test $P = 0{\cdot}005197$**. However, this figure should be multiplied by three, as there are three possible first order associations within Table VI, and we have selected that association showing, by inspection, the highest significance. The true value of P is then 0·0156*.

We have therefore found a significantly higher proportion of subjects of bulbar poliomyelitis showing nystagmus in the second half of the 1949-1950 bulbar series. It is considered that the significant difference observed is due to some change in the observational standards adopted, and a confirmation that the assessment of whether nystagmus is present or absent is subjective to a large extent.

In conclusion, it may be stated that no evidence has been found that tonsillectomy status is associated with nystagmus incidence.

Nucleus Ambiguus Lesions.

Nucleus ambiguus lesions are the commonest cranial nuclear lesions in bulbar poliomyelitis, and are responsible for the characteristic dysphagia, palatal weakness and dysphonia. The writer proposed (1953) that in the tonsillectomized bulbar poliomyelitis subjects the virus of poliomyelitis gained entry to the medulla via the motor nervous pathway from the tonsillar area of the pharynx to the *nucleus ambiguus*. It is therefore worthy of examination whether the proportion of cases of bulbar poliomyelitis showing clinical evidence of a *nucleus ambiguus* lesion differs between the tonsillectomized and non-tonsillectomized classes. The figures for the incidence of such lesions between these classes, for the periods 1947-1948 and 1949-1950 in South Australia, are shown in Table VIII.

Testing the proportions shown in Table VIII, we proceed as follows.

1. If we test the proportion of subjects with a *nucleus ambiguus* lesion in all bulbar cases, 1947-1948 against 1949-1950, the proportions are 36 out of 39 and 21 out of 30; these figures show a significant lack of homogeneity; $P = 0{\cdot}024$*. There is a statistically significant reduction in the proportion of bulbar paralysis subjects showing a *nucleus ambiguus* lesion in the 1949-1950 series as against the 1947-1948 series. The cause of this observed reduction in the proportion is uncertain. Whether it is a chance variation, or due to some variation in the observational standards adopted, or of deeper significance, is not known.

TABLE VIII.

Incidence of Nucleus Ambiguus Lesions in Bulbar Poliomyelitis, for Tonsillectomized and Non-Tonsillectomized Subjects, for 1947–1948 and 1949–1950 in South Australia.

Years.	Tonsillectomized.	Non-Tonsillectomized.	Tonsillectomy Status Not Known.	Totals.
1947–1948 ..	32/35	4/4	—	36/39
1949–1950 ..	11/14	3/5	7/11	21/30
Totals ..	43/49	7/9	7/11	57/69

2. By inspection, the proportions of subjects showing a *nucleus ambiguus* lesion do not differ, on the admittedly small numbers available, between tonsillectomized and non-tonsillectomized classes of bulbar poliomyelitis subjects in South Australia over the period 1947-1950.

SUMMARY.

1. A study has been made of clinical and other features of poliomyelitis, with particular reference to the tonsillectomy status of the bulbar poliomyelitis subjects studied, in South Australia over the period 1947-1950.

2. The clinical features of the nine non-tonsillectomized bulbar cases in South Australia over 1947-1950 available for study are recorded.

3. It is shown that there is a higher incidence of ophthalmoplegias in non-tonsillectomized than in tonsillectomized bulbar cases. This is so for abducens lesions. There is a suggestion ($P = 0{\cdot}07$) that this is also so for oculomotor lesions, but in this latter case the figures available are too small for firm conclusions.

4. The incidences of clinical lesions for other cranial motor nuclei are examined, but no differences were found between tonsillectomized and non-tonsillectomized bulbar classes.

UNIDIGITAL CLUBBING, WITH REPORT OF A CASE.

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ALTHOUGH the literature on the syndrome of hypertrophic pulmonary osteoarthropathy is voluminous, the basic pathogenetic factor concerned, if single, has not yet been agreed upon. Simple finger clubbing is still distinguished from the main syndrome by some authorities, and while a case of unilateral clubbing is unusual, a case of unidigital clubbing is a rarity. Only two case references have been found in the available literature.

Review of the Literature.

Abnormal curvature of the finger nails was described by Hippocrates in the fifth century B.C. in a case of empyema. This physical sign is thus sometimes referred to in the French literature as "Hippocratic fingers", and was neglected until the end of the last century, when von Bamberger (1889) noted its presence associated with pulmonary and cardiac disease. A year later, Marie (1890) introduced the name of hypertrophic pulmonary osteoarthropathy to describe the syndrome of clubbed fingers with periosteal new bone formation and associated arthritic symptoms. More recently, however, some authorities have insisted on the distinction of "simple clubbing" of the finger from the wider syndrome (Pattison, 1951). The former is described as a proliferative change about the terminal phalanges, with no constant osseous change. This is believed by some to be the early stage of hypertrophic osteoarthropathy.

FIGURE I.

Photograph to show clubbing of right index finger compared with left corresponding finger (case reported in this paper).

In 1865 Ogle reported the first case of unidigital clubbing affecting the middle finger of the left hand. In this case the clubbing developed after a blow on the arm which apparently injured the median nerve. The only other cases mentioned in the literature are in a paper by Lebreton (1936). In discussing unidigital clubbing, he suggests that the criteria of decreased angulation of the nail and generalized enlargement of the soft tissues of the terminal phalanx may be found secondary to local disease of the finger tip. He notes cases in which the condition followed local trauma, whitlow, tophaceous rheumatism and Boeck's sarcoidosis involving the terminal phalanx. He also mentions a case associated with congenital brachyphalangia. Contrary to most authors, he sees no reason to distinguish these cases of clubbing secondary to a local digital cause from those associated with remote or systemic disease.

Report of a Case.

A girl, aged eleven years, presented in September, 1953, complaining of an enlarged left index finger. The finger had been noticeably larger in the terminal segment for about three years. Over this period there had been very slow but steady increase in the size of the end of the finger. The patient and her parents were certain that there had been

no antecedent trauma to the arm or affected digit. Investigation revealed that she had suffered from no serious illnesses and that her development had been perfectly normal.

Examination showed the patient to be an intelligent girl, tall and slender for her eleven years. A thorough clinical examination of the body systems revealed no abnormality in the pulmonary and cardio-vascular systems or in the gastro-intestinal tract. The hands were of normal development, with thin, tapering fingers. The terminal segment of the left index finger showed generalized enlargement of the soft tissues, which gave it a bulbous appearance (Figure I). The skin at the base of the nail was more glossy than elsewhere. The nail was increased in area with increased curvature in both directions, but with no signs of ridging. All the remaining digits were normal and symmetrical. The affected finger was slightly pinker in colour than the other digits. There was no evidence of any similar deformity in other members of the family, and there was no family history of tuberculosis. The mother suffers from disseminated sclerosis. Two other children in the family, aged twelve and six years respectively, are normal, healthy children.

A number of investigations were carried out. The urine contained no albumin, sugar or pus. The Mantoux test produced negative results with 1:1000 and 1:100 dilutions. Negative immediate and delayed responses were obtained to the Casoni test. The blood pressure in both arms was 115 millimetres of mercury, systolic, and 70 millimetres, diastolic. A radiograph of the chest showed no sign of a parenchymatous lesion in the lungs. A radiograph of both index fingers (Figure II*a* and II*b*) showed the soft-tissue

FIGURE IIA.

Radiographs to show difference between terminal phalanges of right and left index fingers (case reported in this paper).

swelling of the end of the left index finger, the distal phalanx being broader and longer than its fellow on the opposite side. There was evidence of some sclerosis in the medulla so that clear demarcation between cortex and medulla was lost. There was no change towards spatulation or hypertrophy of the ungual tip of the terminal phalanx.

In view of the enlargement of the terminal section of the digit, associated with both bony and soft-tissue overgrowth, it was decided to give X-ray therapy to the epiphysis of the terminal phalanx. Such therapy, given at a dose level below that required to produce skin erythema, is known to inhibit the growth of epiphyseal cartilage and induce premature epiphyseal closure (Bisgood and Hunt, 1936). At the same time, a degree of *endarteritis obliterans* results from such therapy and would possibly decrease the soft tissue enlargement also. The decision to limit the irradiation to the epiphyseal region was made because of the likelihood of exunguation resulting from an erythema dose to the terminal portion of the digit. Such exunguation is temporary, but has been known to be followed by deformity in the growth of the new nail. Such a result would be unacceptable, in view of the fact that X-ray therapy in this case was given for cosmetic reasons only. A dose of 1000*r* in five days to an area one centimetre square limited to the terminal epiphysis was given, this being considered to be about two-thirds of an erythema dose.

Differential Diagnosis.

Clinical Considerations.

The onset of finger clubbing is, as in this case, usually gradual, but in rare cases may appear within two to six weeks of the antecedent condition (West, 1896). The disease is said to be much more common in males than

FIGURE IIB.

Radiographs to show difference between terminal phalanges of right and left index fingers (case reported in this paper).

in females. In order to make a diagnosis of finger or toe clubbing, there should be a uniform enlargement of the terminal segment, which is usually painless unless it is of acute onset. There is in some cases a feeling of warmth or excessive sweating. If the condition develops during the growing period, there is often an increase in length of the terminal phalanx. The decrease in angle between the proximal portion of the nail and the dorsum of the terminal phalanx is diagnostic. The nail is usually increased in area, and may show longitudinal ridging, increased rate of growth and brittleness.

The "drumstick finger" results from hyperplasia of the soft tissues of the phalanx, which causes a bulbous appearance. The skin over the base of the nail is usually shiny and deeper in colour, and shows slow recovery of colour after pressure.

Radiological Considerations.

In early cases of finger clubbing, it is said that no changes are seen in the bone of the terminal phalanx. In late cases, however, there has been rarely noted an increased spatulation of the ungual process of the terminal phalanx, and in advanced cases osteoporosis appears and may lead to complete absorption of the ungual process (Weems and Brown, 1945). If hypertrophic osteoarthropathy is associated, the bones involved are usually the long bones; but rarely the phalanges also may show periosteal new bone formation. Camp and Scanlon (1948), in their series of cases of idiopathic clubbing, note that the phalanx is of normal length and the metaphysis and

epiphysis are normal. However, the diaphysis is thickened and the ungual tuft atrophic. The radiographic distinction from acromegaly is stressed, in that in the latter disease the diaphysis is slender and the end of the terminal phalanx is enlarged with a hypertrophic ungual tuft.

Pathological Findings.

Pathologically, the only constant change noted in cases of clubbed fingers is tissue œdema and late proliferation of the fibro-elastic tissue of the nail bed. There is sometimes also thickening of the skin and periosteum. Most writers report dilatation of capillaries and increased thickness of the walls of the smaller vessels, and one author notes the formation of new capillaries.

The theories as to the basic causation of the disease have therefore included as factors circulating toxins, nerve lesions, capillary stasis and local anoxia, and lymphatic stasis. Endocrine dysfunction originating in the pituitary, thyroid, parathyroid or gonads has also been blamed. Local anoxia in the tissues of the phalanx is stressed by Lebreton (1936), who thereby is able to correlate the cases of unidigital clubbing in which digital disease impedes the local circulation. Mendlowitz (1942) suggests the importance of increased peripheral blood flow and increased digital arterial pressure in the acquired symmetrical type, but agrees that nerve lesions or lymphatic stasis may explain some cases of unilateral clubbing.

Ætiology.

It is convenient briefly to review the classification of digital clubbing according to the ætiology, as follows: (i) symmetrical, (*a*) hereditary, (*b*) acquired; (ii) unilateral; (iii) unidigital.

Symmetrical Acquired Type.

The symmetrical acquired type is by far the most common and has the following major ætiological factors: pulmonary, cardiac, hepatic and gastro-intestinal.

Pulmonary factors may be any of the following: chronic suppuration (bronchiectasis, pulmonary abscess, empyema); thoracic neoplasms (carcinoma of the bronchus or thymus, reticulosis of the lung or mediastinum, pulmonary hæmangioma and malignant metastases to the lung); pulmonary tuberculosis; chronic pneumonitis (pneumonoconiosis, atelectasis, syphilis, actinomycosis and hydatid disease of the lung).

Cardiac ætiological factors are congenital heart disease with cyanosis, subacute bacterial endocarditis, and congestive heart failure (rarely).

Hepatic factors are cirrhosis (especially Hanot's biliary type), and amyloidosis (rarely).

Gastro-intestinal factors are chronic diarrhœa (dysentery, colitis, enteritis, intestinal tuberculosis, sprue and steatorrhœa), pyloric obstruction, and ascaris infection (rarely).

Miscellaneous factors include primary polycythæmia, syringomyelia (distinct from "*main succulente*"), Raynaud's disease, scleroderma, acrocyanosis and chilblains.

Unilateral Type.

The unilateral type is seen much less commonly, but is found especially secondary to venous compression and thus associated with aneurysmal dilatation of the aortic arch, or of the innominate or subclavian artery. Cases have been reported in which clubbing was associated with brachial arterio-venous aneurysm, venous varicosities of the arm, recurrent subluxation of the shoulder, Pancoast tumour of the lung involving the sympathetic ganglia, and following lymphangitis of the arm. In unilateral clubbing according to Mendlowitz (1942), the finger tip may be longer, shorter or the same length as that on the opposite side.

Unidigital Type.

In the rare cases described of the unidigital type, the ætiological factors noted have been injury to the median nerve, local trauma to the finger, Boeck's sarcoidosis or tophaceous rheumatism. Thompson (1949), with reference to this condition, states: "In most cases the clubbing has succeeded an injury to the arm. The classical example is damage to the median nerve from a crushing injury with subsequent development of unidigital clubbing accompanied by trophic changes." On the other hand, Mendlowitz (1942) states: "Unilateral or unidigital clubbing apparently occurs in association with vascular or vasomotor disease of the corresponding extremity or finger." He quotes Neurath (1932) as stating that bilateral unidigital clubbing, especially of the thumbs, is hereditary.

Hereditary Clubbing.

In this respect we may note that Horsfall (1936) considers that hereditary clubbing differs from the other types in that it does not always appear on all digits, but that it is generally bilateral and symmetrical, and that the thumbs are typically involved. The deformity in these cases is inherited as a dominant Mendelian characteristic, and has been seen associated with other hereditary malformations.

The hereditary type is, of course, constant in its deformity, while the acquired type fluctuates in degree with the severity of the underlying disease and may even disappear. A radiograph of the hand in a case of hereditary clubbing is shown in Figure III.

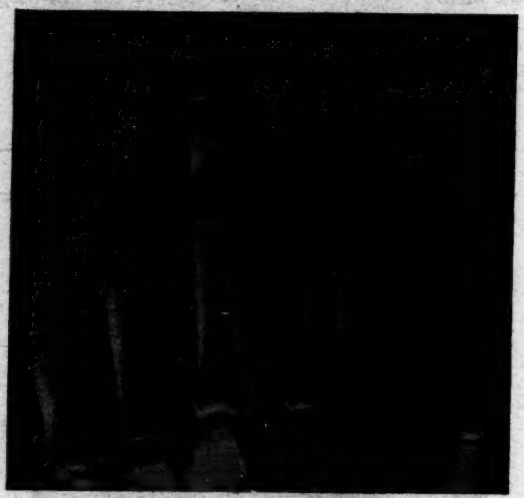

FIGURE III.

Radiograph of one hand in a case of hereditary symmetrical clubbing of all fingers.

Idiopathic Clubbing.

There still remain the cases of idiopathic clubbing in which no underlying cause or hereditary tendency can be shown. In these cases, an unsuspected systemic lesion may show at a later date. On the subject of truly idiopathic clubbing, there have been several reports in the literature of recent years (Camp and Scanlan, 1948; Berk, 1952; and others). Camp and Scanlan (1948) give an excellent review of the literature of chronic idiopathic hypertrophic osteoarthropathy. They mention 21 cases in the literature, and add five cases of their own noted at the Mayo Clinic. They note the condition as occurring predominantly in males at the age of puberty and adolescence, and involving clubbing of the fingers, enlargement of the bones and joints, and coarsening of the skin of the face. The course of the disease is slowly progressive to deformity and disability, and they report that the idiopathic form constitutes 3% to 5% of all cases of osteoarthropathy. They consider that hormonal imbalance at puberty is the exciting cause of the disease in these cases, and differentiate the hereditary type of clubbing, which appears at birth and is occasionally familial (Witherspoon, 1936).

Summary.

The features of interest in this case of unidigital clubbing are that it is the first recorded case of unidigital clubbing with apparently idiopathic development.

The onset at puberty suggests some relationship to the group of idiopathic cases recently described; but these are said to be progressive, symmetrically involving all fingers, and associated with changes in the long bones and joints, skin changes in the face and hormonal imbalance. Our case shows none of these features; but the radiographic appearances are similar to those described by Camp and Scanlan (1948) in that type of case.

The increase in length of the terminal phalanx, which is unusual, led to a decision to give X-ray irradiation to the terminal epiphysis in order to prevent further growth and consequent deformity.

Acknowledgements.

Acknowledgements are due to Dr. J. Hamilton Smith for permission to reproduce the radiograph, to Mr. A. Parry for the photographs and X-ray reproductions, and to Mr. A. Brown, who assisted by translation of foreign language material.

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MYOCARDIAL NECROSIS AND INFARCTION IN RELATION TO EFFORT.

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ON few issues has the medical profession been more divided than it is on the part played by effort in the ætiology of myocardial infarction and necrosis. In view of the enormous importance of the matter in insurance practice, divergence of opinion is unfortunate. It is possible that most of the apparent difference derives from preoccupation with different aspects of the problem, and that on all matters of practical importance there is complete agreement.

Vascular Changes.

Few contend that activity, excessive or otherwise, influences the development of the arterial disease itself. With the possible exception of our own, no occupation produces a significantly higher than average incidence of coronary artery disease, and in the case of doctors, it must be hard to be sure. Doctors are less likely to dismiss lesser coronary symptoms as indigestion, as others so often do. In other walks of life, statistics offer no evidence that laborious work or long hours or the like increases the arterial disease. Coronary atherosclerosis—or atherosis—has long been regarded as but one aspect of that degenerative process which, with aging, spares few organs or tissues of the body—a reminder that man is born to die, and decay is his destiny. More recently it has been suggested that not degeneration but deposition plays the major role, and the importance of diet and other metabolic factors becomes increasingly realized; but such factors are independent of occupational activity. In the development of coronary atherosis, race and heredity cause pronounced differences within any occupational group, and diet may divide us; doings do not.

However, arising out of the arterial disease, there is one quite common happening which perhaps has given rise to most of the disputation—the rapid occlusion of a branch of a coronary artery by thrombosis. Some pathologists maintain that this nearly always originates in a small hæmorrhage just below the surface of the vessel; others warmly dispute this. It is frequently pointed out that the end of this process, the final occlusion, very often occurs during rest in bed—which does not prove very much if the hæmorrhagic or thrombotic process occupies twelve hours or more, as many believe. Efforts have also been made to demonstrate that a rise of blood pressure, such as accompanies exertion, cannot initiate the hæmorrhagic process, if it exists.

It is not suggested that some final pronouncement can now be made, placing all these questions beyond dispute, but rather that this whole matter can be laid aside as a rather captivating academic toy, devoid of any practical importance.

The Myocardium.

With all coronary disease, the thing that matters, the cause of all symptoms, is not what happens to the artery but what happens to the heart muscle, as a result of loss of blood supply. Any examination of this aspect quickly reveals that attention has long been concentrated on the wrong vessel—a legacy from earlier failure to realize the enormous potential capacity of collateral circulation. With a tree, if all the vessels of a branch are occluded at the stem, all that branch will die, every leaf and twig—complete occlusion produces one precise result; but with the occlusion of a coronary artery the result is far otherwise, owing, of course, to a rapidly developing collateral supply. Rapid occlusion will indeed produce an area of myocardial death, or infarction; but within this area by no means all the tissue dies. A ten-day-old infarction exhibits a variety of conditions—healing necrotic areas, healthy muscle, and areas of new infarction (Wessler *et alii*, 1952). Some muscle tissue was killed at the first blow; some was never threatened; much is sick, even near to death, its precarious vitality dependent on a critical balance between collateral supply and metabolic demand. Every day augments supply; but during those critical early days when it is but a trickle, any increase in demand, through effort, may spell doom for an area which was only just holding its own.

Other factors, of course, may disturb the balance. Those who, in serious illness, have experienced the exhaustion that follows an all-over sponge, or other details of the toilet, can appreciate this fact. Increased metabolic demand, through effort, is not the only possible disturbing factor; but a factor it must be, and with the issue so finely in the balance, who can define the degree or duration of increased demand required to turn morbidity to mortification?

Reflection will establish the validity of similar considerations when occlusion is but partial. Loss of calibre from atherosis can proceed so slowly that collateral supply is always ahead of demand and myocardial health is preserved; but it is not always so, and not merely patchy necrosis but frank infarction can and quite frequently does occur without actual occlusion. Different observers have found from one-sixth to one-third of infarctions occurring without occlusion (Dressler, 1944), and it may well be that in non-fatal cases the proportion is much higher. In a study of such infarctions, Master (1950) found evidence of unusual strain in about 30%—rapid depletion of supply through blood loss or emotional con-

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striction, increased demand as from severe and prolonged exertion. However, the real significance of this analysis lies in the fact that in 70% no such factor was found. Anatomical changes in the vessel, short of occlusion, reduced flow too rapidly for the life-saving collateral supply to become effective. The situation is just the same as with complete occlusion. Within the infarct there will be areas whose vitality is in the balance—or indeed most of the area may be critically sick but not beyond hope of recovery. With myocardial integrity balanced on a knife-edge, how small or how brief an increase in relative ischæmia will suffice to carry morbidity beyond the point of no return?

Effort as a Factor.

That exertion can produce at least a brief increase of relative ischæmia is, of course, a commonplace. Have we, on clinical grounds, reason to believe that, in circumstances such as described, effort can and does produce infarction, or some part of an infarction, or at least myocardial necrosis?

We are all familiar with the patient in whom some perhaps not unusual effort produces severe anginal pain which soon subsides with rest, but is followed by a greatly increased sedimentation rate and perhaps, months later, by alteration of *T* waves. Can we doubt that some necrosis has occurred? There is in fact an abundance of literature on the subject of what is customarily designated "coronary insufficiency", a term used to cover all grades of painful episodes more severe than typical angina of effort, but short of catastrophic infarction (Blumgart *et alii*, 1941; Dressler, 1944; Littman and Barr, 1952). All such episodes are considered to result in some degree of myocardial necrosis, and ultimate post-mortem findings consistently support this view. Not all such episodes result from effort. Angina of rest indicates arrival at that delicate balance which has been described, when even the natural variations of blood pressure, or constriction of coronary arteries by emotion, cold, tobacco, or other factors, produce painful ischæmia in just the same way as effort may. Dressler concluded that angina of effort, or brief attacks of angina of rest, might be significant symptoms of ischæmic myocardial necrosis—which is in keeping with the fact that, in cases of angina, multiple infarction is the commonest post-mortem finding (Zoll *et alii*, 1951). In brief, while there is no suggestion that all myocardial necrosis results from effort, all our experience reveals the closest association between cardiac pain produced by effort and signs of myocardial necrosis.

It is sometimes objected that, while effort may indeed produce painful myocardial ischæmia, the pain will terminate the effort before any permanent myocardial damage can result; and it may be pointed out that a duration of twenty minutes is required for coronary ligation to produce infarction in a healthy canine heart. As to that, the situation is, of course, vastly different when that delicate state has developed at which, in one or more areas, myocardial survival trembles in the balance. Apart from this, however, vasomotor reactions, provoked by some exertion, may maintain ischæmia for quite a time after the exertion has ceased. The fall of blood pressure on cessation of effort is normally brief, with little disturbance of mean pressure; but Fraser and Chapman (1954), in investigating these reactions, found that in some cases systolic, diastolic and mean pressures remained below their normal resting values for more than five minutes. That vasomotor reactions following effort are not always perfectly controlled is borne out by experience. In the condition of neuro-circulatory asthenia, at one time referred to as "D.A.H.", profound collapse following effort may continue for a quarter of an hour or longer; but indeed there are all degrees of excessive vasomotor reaction. Frequently a man who has helped to push a motor-car, or sprinted for a tram, next minute feels "all in", looks pale, and causes a good deal of concern for perhaps ten minutes or so, and is then himself again. Who can doubt that, during the collapse, coronary circulation was greatly depressed? How serious the consequences could be if an area of myocardium was at the critical state! Indeed, is it not probable that it is in these circumstances that perhaps infarction, perhaps sudden death, often ensues as the result of excessive vasomotor reaction provoked by effort.

There may be other factors causing prolonged ischæmia after effort. Rapaport *et alii* (1952), in a review of experiments on the lower limbs, cited cases showing evidence of vascular spasm lasting up to fourteen minutes after exercise, even when vasoconstrictor nerves had previously been severed. It is known that myocardial necrosis provokes vascular spasm in the vicinity. May there not be some similar effect from even temporary relative ischæmia of painful degree, resulting from effort? At all events, in the case of vasomotor reactions there is no doubt. Increased myocardial ischæmia can continue for some time after exertion has ceased, owing to excessive vasomotor reactions which the exertion has provoked.

Whenever it becomes a question of degree—including, in this case, duration—there is one line of reasoning which often makes the issue very clear. If the strain or effort did not do the damage, then it happened of itself. With the thing in that state, would not the slightest added strain produce the catastrophe?

Myocardial necrosis covers a wide range. At times it will appear that, even if a specific effort has produced some necrosis, as part of the over-all picture the effect must be very slight. We should remember that it is not our province, as medical advisers, to determine compensability when that is the issue. We may express the opinion that one particular episode could have very little effect on incapacity, even if some permanent myocardial damage did result. We may also be asked to consider whether the same thing probably happened many times, with a quite appreciable cumulative effect. One duty, however, is plain. If we think that any permanent damage at all could have resulted from a specific effort, we must say so, and the question for us to answer is not "Can I be sure the effort did it?", but "Can I be sure it did not?".

The Issue Stated.

Myocardial necrosis and infarction can occur without coronary thrombosis, but even when thrombosis is known to have occurred, the extent of infarction or necrosis is by no means determined on the occurrence of thrombosis. Both during the development of thrombosis, and when it is complete, other factors, influencing the balance between collateral supply and metabolic demand, will determine the amount of ultimate necrosis and can thus influence the consequences, even when death ensues. Without thrombosis, the imminence of infarction may be indicated by the onset of angina of effort, by an increase in the ease with which angina of effort is produced, and, above all, by the appearance of angina of rest. Given time, the development of collateral circulation may avert the occurrence of infarction, or minimize its degree. In all these cases, and in many others in which infarction occurs unheralded, there is a critical period when almost any increase of relative ischæmia will cause infarction, or increase its degree. Effort produces just such an increase of relative ischæmia, which may continue after the effort ceases. With a frequency which, if coincidental, is truly remarkable, effort and infarction or at least necrosis are found in the closest temporal relationship. Can anyone be sure that the relationship is not causal?

Summary.

The relation of myocardial necrosis and infarction to effort is discussed.

It is suggested that attention be concentrated on the myocardium, since changes in the coronary arteries do not determine the final result.

The question is asked whether, in the presence of a close temporal relationship, it is possible to be sure that the effort did not influence the outcome.

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TENNIS HEEL.

By J. WOOLNOUGH, M.B., B.S.,
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PLANTAR FASCIITIS or plantar fibrositis is characterized by a tender spot under one heel, towards the front and often the medial side of the "pad" of the heel. It is usually chronic or recurrent in nature, causing pain on walking or standing, though there is no pain when the foot is not weight-bearing, except perhaps a dull ache at the end of a working day. However, deep tenderness is constant throughout, and may be confused with little else except a "stone bruise" (small hæmatoma or abscess) in the same situation.

The main purposes of the present communication are to supply the condition with a usable or common name and to indicate a new line of treatment.

The name "tennis heel" was chosen for the following reasons: (i) The case in which the treatment to be described was first used appeared to have arisen from an undue amount of tennis played on hard (asphalt) courts. (ii) There seem to be certain resemblances to "tennis elbow"—namely (*a*) the pathology may be akin; (*b*) the course and duration are often similar; (*c*) like "tennis elbow", the condition may be caused by many things besides tennis. On this score I should like to add that I wish no harm to tennis, perhaps the best of outdoor sports.

Pathology.

The lesion occurs at the posterior insertion of the plantar fascia and the plantar muscles into the anterior edge of the calcanean tubercles, medial or lateral, but more commonly the medial. The condition may be ascribed to aseptic or traumatic inflammation which, if prolonged, may go on to bone formation—calcanean spur.

Little has been written about it; but some text-books blame the causation on "focal sepsis, gout, rheumatism, etc.". *These seem to me to be the ætiological rubbish-bin* for conditions of unknown origin and unsatisfactory treatment.

The ætiology is surely trauma—repeated trauma; for this is one of the points of maximum strain in the body, where *great longitudinal tension in the plantar soft structures* may (in walking) alternate with the direct weight of the whole of the body applied to the same point. Unaccustomed use of the feet on hard surfaces, perhaps with *light* footwear, by a young or middle-aged person may be the *precipitating factor. Even so, the condition* is usually unilateral.

Treatment.

Avoidance of weight-bearing on the affected foot would presumably cure tennis heel. How long this would take *is not known, because nobody has ever tried it long* enough for complete cure.

Of text-book treatments, the only ones of value are the following: (i) insertion of a sponge rubber pad *inside* the shoe, under the heel; (ii) building up the heels of the *shoes half an inch; (iii) the use of a solid rubber heel* on the shoe; (iv) the oral administration of cortisone (used with benefit).

A treatment which has not, to my knowledge, been described before, involves the use of adhesive strapping (non-elastic) to support the longitudinal arch and relieve the tension on the plantar soft structures in walking. Applied each morning and discarded each night, this support surprisingly affords almost complete relief of symptoms if the daily activities are restricted to walking or standing. Presumably bony spur formation could reliably be prevented. But the duration of the condition is not altered, and treatment may be needed for many months before the tender spot has gone.

The method is as follows. A good brand of sticking plaster two inches wide is used. Some reels are not quite so good as others—only the best will work. The foot should be warm, clean and dry. The strapping is applied first to the ball of the foot, under the medial three metatarsal heads, right up to the roots of the toes. It is held here till it gets a firm grip. Then the foot is plantar flexed a little, as this shortens the longitudinal arch, and the strapping is drawn on to the undersurface of the heel and round on to the back of the heel for one to two inches, and cut off there. It should then form the flat deck of the bridge whose arch is the longitudinal arch of the foot.

It is surprising how strong a grip good strapping can maintain. After half an hour the foot is quite comfortable and forgotten. At the end of the day the strapping is usually flapping rather limply, but even then seems to be of some help.

Summary.

The name "tennis heel" is suggested for plantar fasciitis. A new treatment for this painful condition is described, involving the daily support of the longitudinal arch of the foot with adhesive plaster.

THE ECTOPIC URETER.

By DOUGLAS COHEN,
Sydney.

THE majority of cases of ureteral ectopia are reported because of the supposed rarity of the condition. Indeed, Alldred and Higgins (1951) commence their excellent article on the subject with this very remark. However, the occurrence of five cases in the last two years at the Royal Alexandra Hospital for Children leads one to suspect that the condition may not be as rare as has been thought. If the condition is not kept in mind it will not be diagnosed, yet it has an almost pathognomonic symptomatology, and only the simplest of tests, which can be performed by any practitioner, are necessary to confirm the diagnosis. Relief of symptoms by operation is immediate and permanent, and *the gratefulness of these little patients for* the physical and psychological relief they experience has to be seen to be believed. They are transformed from miserable social outcasts into happy normal children. They usually have a long history of medical treatment for their *"enuresis" and have been scolded and penalized for* their "non-cooperation". The purpose of this article is to outline clearly the symptomatology and methods of diagnosis of the condition so that it may be treated *with a minimum of* delay and inconvenience to the unfortunate sufferer.

The first recorded case of ectopic ureter that I have been able to trace was reported by Civiale in 1843. This was one of unilateral duplication with an opening into the posterior part of the urethra. *Increasing numbers of these* cases were reported until Eisendrath was able to list a *total of 225 collected cases* up to 1934. Burford *et alii* (1949) reported a collected series of 425 cases, with complete statistics on 404 of these. The reported incidence *is approximately one in 80,000; the actual figure is likely* to be higher than this, since many cases go undiagnosed for a long time, and many males with this condition are symptom free. *The females outnumber the males by about* five to one, according to the most recent series. Most cases

are seen in children in the first decade, and 90% are discovered by the time the subject reaches the age of thirty years. There seems to be an equal incidence as to the side involved.

In the female the vast majority of ectopic ureters open into the urethra (33%), the vestibule (34%) or the vagina (22%); 7% open in the bladder below the normal site but *above the internal sphincter, and are not productive of* symptoms. Very rarely the site of the ectopic opening *may be the uterus, the cervix, the upper part of the vagina* or the rectum. It is difficult to explain this rare group on morphological grounds.

In the male, in most cases the opening is into the posterior part of the urethra or the bladder below the normal site. None of these are productive of enuresis as the outflow can be voluntarily controlled.

The following types of abnormality are met with *(Burford et alii, 1949)*:

Single ureter with ectopic opening:	
Unilateral	16%
Bilateral	3%
Unilateral double ureter:	
Ectopic ureter from upper pelvis	89%
Ectopic ureter from lower pelvis	1·5%
Both ureters ectopic	1%
Type not stated	19%
Bilateral double ureters:	
Unilateral ectopic opening	9%
Bilateral ectopic opening	5%
Congenital solitary kidney with ectopic opening	3%
Horseshoe kidney with ectopic opening	0·5%
Type not stated	3%

Embryology.

The human pronephros has usually completely degen*erated in embryos of five millimetres, but its duct, destined* to become the mesonephric or Wolffian duct, reaches the cloaca in embryos of about four millimetres. Between the four and five millimetre stages—during the fifth week of

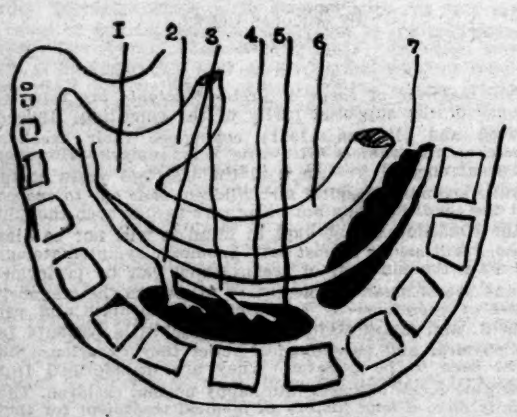

FIGURE I.

Diagrammatic representation of the caudal portion of a five millimetre embryo, in which there is a complete duplication of the ureteric bud; 1, cloaca; 2, bladder; 3, ureteric buds; 4, Wolffian duct; 5, metanephrogenic cap; 6, hindgut; 7, mesonephros.

intrauterine life—a hollow outgrowth, the ureteric bud, arises from the postero-medial wall of the Wolffian duct near its junction with the cloaca. This bud grows dorsally, and at the same time its origin migrates to the postero-lateral aspect of the Wolffian duct, where the cranial aspect of the bud ultimately comes into contact with the metanephrogenic cap to form the definitive kidney, of which the ureteric bud forms the collecting system. The enlarged cranial end becomes marked off into major calyces and gives rise by subdivision into generations of collecting tubules, the second, third and fourth generations being absorbed to form the minor calyces. In the 10-millimetre embryo the Wolffian duct has become absorbed into the *base of the definitive bladder in such a manner that it* and the ureter have acquired separate orifices. With the further growth of the bladder the ureteric orifice is carried upwards and laterally, while *the orifice of the Wolffian* duct, destined to form the ejaculatory duct in the male and Gartner's duct in the female, becomes displaced caudally. By the 13-millimetre stage—during the seventh week of intrauterine life—the cranio-lateral migration of *the ureter is complete.*

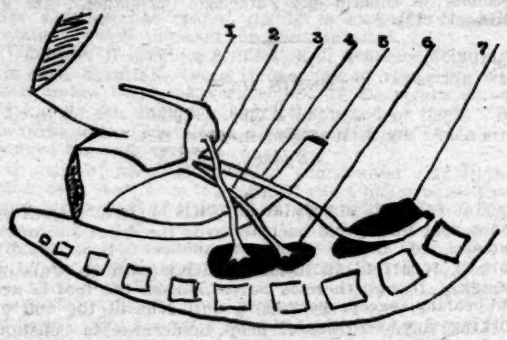

FIGURE II.

Diagrammatic representation of a 15 millimetre embryo, showing complete duplication of the ureteric bud. Migration of the lower ureter is almost complete, but the upper ureter has been carried down with the Wolffian duct. 1, bladder; 2, lower ureter; 3, upper ureter; 4, Wolffian duct; 5, hindgut; 6, metanephrogenic cap; 7, mesonephros.

From the foregoing facts it can be readily seen that incomplete duplication of the ureter can be explained only by a splitting of the ureteric bud from above downwards, which may progress to any point in the length of the ureter. Complete duplication of the ureter can be explained by a complete splitting of the ureteric bud in its whole length, or by the development of two ureteric buds from the Wolffian duct. The final disposition of the two ureteric orifices in complete duplication of the ureter is determined by the incorporation of the lower end of the Wolffian duct in the definitive trigone and the upward and lateral migration of the ureteric orifices. Since the lower ureteric bud, communicating with the lower segment of the kidney, is drawn into the bladder first and immediately commences its cranio-lateral migration, its definitive position will be higher and more lateral than the upper bud which, reaching the bladder later, has less time for its migration, depending upon the time between the liberation of the two ureteric buds into the bladder. If the interval is sufficiently long, the upper bud will never migrate at all, but will be carried down to open with, or close to, the lower end of the Wolffian duct. Occasionally cranio-lateral shift of the upper bud is faulty and it is carried down with the Wolffian duct, but this is exceptional. As a rule the lower ureteric bud crosses in front of the upper one to open at a higher and more lateral level.

Pathology.

The kidney, or portion of the kidney, connected with the ectopic ureter frequently shows pathological changes. It may be hypoplastic, cystic or hydronephrotic, but in some cases it is perfectly healthy renal parenchyma. The practical point is that abnormal, poorly secreting renal tissue will fail to throw any shadow on excretion pyelography. Other concomitant abnormalities in the uro-genital tract are not infrequently associated with ectopic ureter. The ectopic ureter itself is almost constantly abnormal. It is dilated and tortuous, regardless of whether there is any distal obstruction or not. This is probably due, as Campbell (1937) suggests, to a failure of development of the neuro-muscular components of the wall of the ureter.

There may be strictures and valvular formations present at varying levels in the wall of the ectopic ureter. Needless to say, infection of the urine contained in the ectopic ureter supervenes in virtually every case, usually at an early stage. This results in a superadded pyelonephritis in the involved kidney tissue and inflammatory infiltration and fibrosis in the thin ureteric wall.

The orifice of the ectopic ureter may be difficult or easy to find, according to its position and size. Not infrequently it is stenosed and will not admit the smallest probe or catheter.

Clinical Features.

It is rare for the condition to be diagnosed in the male, as incontinence of urine is not a feature, since the ectopic orifice is above the external urinary sphincter. If it is productive of symptoms at all, they will be recurring bouts of urinary infection, the true nature of which may be revealed by full urinary investigation. However, it is rare for the condition to be diagnosed in males during life.

It is in the female child that one meets with the characteristic symptom-complex that is so suggestive of the condition. The story that can almost invariably be elicited is one of dissociated micturition. That is, the child passes urine in normal amounts and at regular intervals and has normal bladder sensation, but in addition incontinence of urine is present. The incontinence may be worse in the daytime than at night, when the child is lying down, the so-called "vertical incontinence"; or it may be most noticeable immediately after micturition. In addition, there is usually a history of recurrent bouts of pyelitis. These unfortunate children have often been treated for their "enuresis" on several occasions without improvement. The perineum becomes irritated and reddened owing to the constant soiling, and the physical discomfort and social ostracism occasioned by the condition lead to profound emotional changes.

Physical Examination.

Although in some older children it is possible to carry out a thorough inspection of the perineum in the consulting room, it is found that in the majority of cases a general anæsthetic is required to allow of more than a superficial examination. There are numerous pits normally found in the vicinity of the urethral orifice which may conceal an ectopic orifice and which have to be probed to determine whether or not they are the source of the abnormal efflux.

If, therefore, general examination and as thorough an inspection of the vulva as is possible without an anæsthetic reveal no abnormality, it is best to admit the child to hospital and conduct the further investigation there.

One simple test will at once serve to demonstrate whether there is indeed an ectopic opening. A weak solution of dye, such as 0·4% indigo-carmine solution, is run into the bladder and serves to colour the urine contained therein. A vulval pad is applied and the child is allowed to walk about and is instructed to refrain from passing urine. The pad is inspected in half an hour, and if it is moist with colourless urine, then the source of the efflux cannot be the bladder. This is confirmed by then getting the child to pass the coloured urine contained in the bladder. If this test gives a positive result, it remains only to determine the site of the ectopic orifice and the side from which the urine comes. An excretion pyelogram may show an abnormal dye shadow above one or both kidneys, but in most cases the concentrating power of the abnormal renal segment is so poor that nothing abnormal is seen. There may be downward displacement of one or other kidney, or the presence of the dilated ectopic ureter may displace the kidney further from the mid-line than usual. However, the important point is that a normal excretion pyelogram is of no significance whatever and does not rule out the diagnosis of ectopic ureter, which has already been established by the dye test. Microscopic and cultural examination of the urine will usually reveal the presence of a coliform infection, which should be suitably controlled at this stage.

Under general anæsthesia a careful search can then be made for the orifice of the ectopic ureter. Most commonly it is situated just behind and to one side of the urethral orifice, but not uncommonly it opens into the urethra or onto the anterior vaginal wall. If it can be located, an attempt should be made to catheterize it; a ureteric catheter stiffened with tonsil wire is useful for this purpose. It may be difficult or even impossible to catheterize the orifice, as it is often stenosed; but if this can be done, sodium iodide solution can be injected, and the course of the ectopic ureter accurately delineated. At the same time cystoscopy and retrograde pyelography can be performed, and the presence of two normally situated ureteric orifices confirmed.

Treatment.

Various operative procedures have been recommended, from simple ligation of the ectopic ureter to complete excision of the abnormal renal segment, together with as much of the ectopic ureter as possible. It is thought that this latter procedure is the one of choice in most cases. The kidney is exposed in the loin, and in most cases the abnormal renal segment is found perched on top of the kidney and clearly demarcated from it. It receives its own leash of vessels, which can be secured, when the ensuing congestion of the abnormal segment serves further to delineate its extent. It is dissected away from the kidney and the dilated ectopic ureter is traced down as far as possible. An oblique extraperitoneal iliac incision then enables the ectopic ureter to be traced well down into the pelvis, where it is usually found to become appreciably narrower and can be conveniently transected. Occasionally it is found that the ectopic ureter arises from the larger portion of the kidney on that side. If this renal tissue functions reasonably well, one may consider anastomosing the ectopic ureter to the lower renal pelvis and then removing its distal portion. Simple ligation of the ectopic ureter is bad surgical practice, as the proximal portion will probably undergo further dilatation and become infected. In the same way, if the bulk of the ectopic ureter is left it may well require removal subsequently.

Reports of Cases.

A, a female child, had developed normally, but despite this she had never had urinary control. She had normal bladder sensation and could pass from one to five ounces of urine at intervals during the day but, despite this, urine began to dribble away again five to ten minutes after micturition. About every two months she suffered from recurrent bouts of urinary infection. Her bowel control was perfect. General examination revealed her to be a healthy child with no abnormality detectable in any system. There was a continuous seepage of urine from the vulva, which was somewhat reddened.

She was admitted to hospital at the age of two years. Examination under anæsthesia revealed urine to be seeping from a rather patulous urethra, but no ectopic orifice could be detected. Excretion pyelography revealed a functioning kidney on each side with a normal pelvi-calyceal system. Indigo-carmine (0·4%) was introduced into the bladder, but was expelled almost immediately in a few small gushes.

The child was discharged from hospital and kept under observation at the out-patient department. As her incontinence persisted, and her story was so suggestive of an ectopic ureter, she was readmitted to hospital at the age of three years for further investigation.

On this occasion, when indigo-carmine was introduced into the bladder it was retained, but clear urine continued to drain away. In spite of this, the site of the ectopic orifice could not be found. Cystoscopy and retrograde pyelography showed a normally placed ureteric orifice on each side, and a normal ureter and pelvi-calyceal system, which appeared to be situated further from the mid-line than usual. Excretion pyelography confirmed these findings, but failed to reveal any additional dye shadow above either kidney. It was decided to explore the right kidney, as this was furthest from the mid-line. The kidney was approached through the usual lumbar incision, and an abnormal segment of renal tissue was found at the upper pole, fairly well demarcated from the remainder of the kidney and with its own blood supply. When this was tied off, the abnormal segment became congested and clearly marked off from the remainder of the kidney. The ectopic ureter was grossly dilated and was pushing the kidney laterally, thus accounting for the radiological findings. The ectopic ureter and attached renal tissue were dissected free and followed down into the pelvis, an inguinal extraperitoneal

incision being utilized. The ectopic ureter was transected low down in the pelvis and removed.

The child continued to suffer from incontinence of urine, although the mother stated that she "only seemed to be half as bad as previously". She was therefore readmitted to hospital and exploration of the left side revealed a similar condition to be present. On this occasion there was considerable inflammatory reaction around the ureter, making the dissection much more difficult, so that the ectopic ureter was cut across about two inches below the kidney and the pelvic portion was left and may possibly require removal later. The child has been completely "dry" since the second operation.

B, a female patient, was aged ten years. This case has been reported already (Cohen, 1953). There was a unilateral ectopic ureter running from an abnormal renal segment situated at the upper pole of the right kidney.

C, a female patient, aged nine years, was in the care of Dr. E. Stuckey. She had suffered from persistent leakage of urine since birth when upright. Incontinence was relieved when she was lying down. She was able to empty the bladder in the normal way and had normal bladder sensation, but in spite of this, leakage of urine was present immediately after micturition. The leakage was slight but constant while she was standing, requiring a change of underclothing three or four times daily. She had previously been investigated by a urologist when aged five years, but no abnormality was discovered at that time.

On examination of the patient, there was irritation of the skin around the vulva, but no other abnormality was discovered on routine clinical examination. An excretion pyelogram was reported as having normal appearances, but when the films were reviewed subsequently it was shown that very faint evidence of a hydronephrotic segment at the upper pole of the left kidney was present. Examination of the patient under anæsthesia disclosed an ectopic ureteric orifice just within the vagina on its anterior wall. This was catheterized and sodium iodide solution was injected, a dilated and tortuous ureter leading to a grossly hydronephrotic pelvi-calyceal system at the upper pole of the left kidney being revealed. Cystoscopy and retrograde pyelography revealed two normally situated ureteric orifices and no other abnormality, except that the upper calyx on the left side was not filled from the left ureter.

At operation the presence of an ectopic ureter communicating with the upper pole of the left kidney was confirmed. This segment was supplied by a leash of vessels slightly larger than the main renal vessels themselves. When they were tied, the upper two-thirds of the kidney became congested, so that heminephrectomy was performed. Convalescence was uncomplicated, and the child has been "dry" ever since.

D, a female patient, aged two and a half years, was in the care of Dr. E. Goulston and Dr. C. J. Gibson. She had been incontinent of urine since birth, both by day and by night. There appeared to be a constant steady dribble of urine, but in spite of this she passed normal amounts of urine about six times a day and appeared to have normal bladder sensation. Normal bowel control was present. Physical examination revealed her to be a well-nourished child with no apparent abnormality. The urethral orifice appeared normal and no ectopic orifice was evident. There was a small amount of urine continually escaping from the vagina and moistening the perineum. Excretion pyelography revealed a bifid kidney on the right side, there being no evidence of double ureters, the point of junction being at the level of the lower part of the pelvis. On the left side there appeared to be a double kidney with a dilated upper calyceal system. Two ureters were present, but their point of junction could not be made out. Retrograde pyelography revealed two normally placed ureteric orifices. It was possible to fill only the lower left renal pelvis from the bladder, but complete filling was obtained on the right side. Blue dye was run into the bladder and a pad applied to the vulva. This soon became soaked with colourless urine.

The left kidney was explored and found to have two ureters, the one to the upper pole being the larger. Several vessels to the upper pole were ligated, and the upper half of the kidney was removed together with the upper ureter, which was traced as far as the pelvic brim. The child had an uncomplicated convalescence and has been "dry" ever since.

E, a female patient, aged five years, was in the care of Dr. J. Steigrad. Since birth the child had passed urine four or five times daily. She had no history of enuresis and no difficulty in initiating the act of micturition, but after apparent cessation urine would continue to flow intermittently, drop by drop, for about ten minutes. Clinical examination revealed no abnormality. Indigo-carmine was run into the bladder and a vulval pad was applied. This remained perfectly dry until the child was allowed to pass the blue urine from the bladder. The pad was then reapplied and became moistened with colourless urine. The intravenous injection of indigo-carmine resulted in blue coloration of both the urine passed naturally and the subsequent efflux, which appeared to be coming from the urinary meatus. An excretion pyelogram was reported on as follows:

> There is a bifid right kidney and two ureters. The lower pelvi-calyceal system is drained by a ureter which is of normal size throughout. The upper part of the right kidney is drained by a dilated ureter which appears to empty below the bladder. The left urinary tract and bladder are within normal limits.

The right kidney was explored by an extended lumbar incision and a bifid kidney was found, the upper ureter being dilated. The upper portion of the kidney was supplied by a separate leash of vesels, and when these were ligated about half the kidney became congested. Heminephrectomy was performed, the ectopic ureter being followed down into the pelvis, where it was ligated and transected.

Convalescence was uneventful, and the child has had no recurrence of symptoms since operation.

Summary.

The incidence, embryology and pathology of ectopic ureter are described. It is suggested that the condition is probably not as rare as has been supposed, and that many cases may be missed.

The clinical features are discussed. A history of "dissociated micturition" can usually be elicited and is strongly suggestive of the condition. The dye test provides a simple method of establishing the diagnosis.

The treatment of the condition is operative, and partial nephroureterectomy is usually the operation of choice.

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KERNICTERUS, WITH SPECIAL REFERENCE TO DOUBLE EXSANGUINATION TRANSFUSIONS.

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KERNICTERUS is a condition seen in the neonatal brain due to deposition of bile pigment in nerve cells, myelin sheaths and interstitial tissue in localized parts of the cerebellum, mid-brain and brain stem; it is not usually localized to the nuclei of the brain, although the name originally implied this. The areas most commonly involved are the *globus pallidus* and putamen, the *cornu ammonis* of the hippocampus, the nuclei of the pons and medulla in the floor of the fourth ventricle, the mesial portions of the thalamus, the flocculus and dentate nucleus of the cerebellum and the hypothalamic area, and sometimes the ependymal lining of the ventricles.

Ætiology.

Originally it was considered that kernicterus was a specific entity associated with hæmolytic disease of the newborn, but Zuelzer and Mudgett (1950) showed that a

number of neonatal diseases associated with jaundice, such as prematurity, severe infections, diarrhœal diseases, hæmorrhage and maternal diabetes, might be encountered in cases of kernicterus. Furthermore, histologically identical lesions without the bile staining have been demonstrated in non-jaundiced premature infants who had suffered from severe anoxia (Govan and Scott, 1953).

It seems, therefore, that certain areas of the neonatal brain are susceptible to damage whether from anoxia or from toxic agents. Experimentally it is said to be impossible to produce yellow discoloration of the brain by the injection of either directly or indirectly reacting bilirubin without first damaging the nerve cells by X radiation or some other means; but whether this is also necessary in the human brain is open to doubt.

Hsia *et alii* (1952) have shown that the incidence of kernicterus rises in direct proportion to the rise in serum bilirubin level, but it has not yet been definitely demonstrated that the high level of bilirubin has a direct toxic effect on brain cells, or whether some additional factor such as cell damage or altered permeability of the blood-brain barrier is a prerequisite.

Boorman and Dodd (1943) demonstrated in Rh-positive subjects that not only erythrocytes but other organs contained Rh-specific substance, capable of neutralizing Rh antibodies.

Van Bolhuis (1950) found that a large number of Rh-positive persons had receptor substances on their erythrocytes, but only a small proportion of these persons (probably about 10%) also possessed Rh antigens or receptors in the placenta and other organs including the brain. These receptors are capable of fixing the maternal antagonistic antibody and may thus effect cell damage. He also demonstrated in two cases of kernicterus that receptor substances were found in greater concentration in the *corpus striatum* than in other parts of the brain. Clinically, his findings would seem to be corroborated by the fact that approximately 10% of neonates afflicted with hæmolytic disease develop kernicterus. It may be that the ability to produce these specific receptor substances is genetically acquired from the parents, and this would provide a reasonable explanation for the hereditary tendency frequently recorded in kernicterus.

Furthermore, it has been recognized for many years that the bile pigment found in the brain in nuclear jaundice differed from that discoverable in other cases of jaundice; for instance, icterus of the brain does not appear in adults with pronounced obstructive jaundice, or in congenital atresia of the bile ducts.

Schmorl (1904) noted that the organs of newborn infants that had died of nuclear jaundice remained yellow in formalin, whereas in other cases of neonatal jaundice the organs become green.

Zuelzer and Mudgett (1950) also noted that in the organs of infants that had died of kernicterus, after fixation in formaldehyde, the pigment was bright orange-yellow, and after exposure to light and alcohol it tended to fade instead of turning green, as did the organs in other cases of jaundice. The explanation of this phenomenon has been supplied by Claireaux *et alii* (1953), who demonstrated by the method of partition chromatography that the pigment found in the brains of infants who had died of kernicterus was indeed indirectly reacting protein-bound bilirubin, which retains a stable yellow coloration and is not oxidized to the green biliverdin form, as occurs in the less stable direct-reacting pigment. The total circulating bile pigment may reach high levels—for example, up to 50 milligrammes per 100 millilitres or more; but if the indirectly reacting bilirubin content is low, kernicterus does not develop. Furthermore, they have shown that brain tissue has a selective attraction for bilirubin, because it contains a bilirubin-retaining lipid of undiscovered constitution which has no affinity for the directly reacting bile pigment. The conversion of bilirubin to directly reacting pigment tends to protect the neonatal brain from icterus; this change is made by the liver, probably in the polygonal glandular cells surrounding the bile capillaries. Therefore reduced liver function may contribute to the development of brain icterus in the neonate suffering from hæmolytic disease, prematurity or certain toxic and infective conditions.

Himwich and Nahum (1932) have made the observation that in week-old puppies the medulla and mid-brain have a higher oxygen consumption than has the cerebral cortex, but the reverse relationship holds true in adult dogs, the increased oxygen consumption of the higher centres in grown animals being due presumably to their having acquired greater functional importance and assumed a position of dominance over the lower centres.

Reversibility.

The four cases presented below are significant, because they demonstrate that the changes occurring in the nervous tissue of infants suffering from kernicterus are reversible probably up to a period of as long as forty-eight hours.

At present there is no way to determine whether these changes in the nervous tissue are brought about by anoxia, or as a result of the products of an antigen-antibody reaction, or by the direct toxic action of bilirubin, or by a combination of all these factors. However, these changes can be reversed and clinical normality restored by the use of repeated exsanguination transfusions. It is quite certain, from years of previous experience, that in the absence of such therapy infants afflicted in this way usually die on the fifth day of life, or if they survive they almost invariably retain severe sequelæ, such as spasticity of the limbs, mental retardation, deafness *et cetera*.

CASE I.—A, a female, was born on February 5, 1954, the fourth child of an Rh-negative mother and an Rh-positive father. All her siblings had been normal and not jaundiced in the neonatal period. This child was born at full term, the labour was normal, and the birth weight was six pounds four ounces. Jaundice developed five hours after birth, the liver and spleen were enlarged, and the hæmoglobin value of the capillary blood was 9·1 grammes *per centum* (64%). The baby's blood group was O(IV), Rh-positive, and her blood gave a strong direct reaction with Coombs serum. At the age of eighteen hours she was admitted to the Royal Children's Hospital, Melbourne; there an exsanguination transfusion of 760 millilitres of blood was undertaken at the age of twenty-three hours. After this the jaundice persisted and deepened in intensity.

On the fifth day of life twitching of the limbs and face commenced, and scattered petechiæ were observed on the trunk and limbs. Calcium gluconate was administered and a lumbar puncture revealed xanthochromic cerebro-spinal fluid not under greatly increased pressure, and there was no cellular increase. As some umbilical sepsis was present, a blood culture was attempted, but no growth was obtained. The hæmoglobin value of the blood was 13·5 grammes *per centum* (87%). Three convulsions occurred.

Forty-eight hours after the onset of neurological signs, on the seventh day of life, the infant exhibited clenched fists, rigid arms and legs and rolling eyes, and jaundice was intense. A second exsanguination transfusion was then given through the saphenous route; after 656 millilitres of blood had been exchanged the infant collapsed and was eventually revived with "Coramine" and oxygen. The transfusion was discontinued.

Next day the infant no longer exhibited any neurological signs, although some jaundice persisted; a hæmoglobin estimation yielded a result of 15 grammes *per centum* (100%). The infant was discharged home, well, on the twenty-third day.

Close observation has been made in the eight months subsequent to her discharge from hospital, but as yet there is no evidence of neurological disease, the cranial dimensions are normal, and the infant is successfully passing her "milestones" at a slightly precocious rate.

CASE II.—B was born on April 25, 1954, the second child of an Rh-negative mother and an Rh-positive father; her elder sibling was normal at birth. She was delivered normally at term and her birth weight was seven pounds eight ounces. Four hours after birth she developed jaundice and œdema, and was admitted to the Queen Victoria Memorial Hospital, Melbourne. An obvious degree of jaundice, hepatosplenomegaly and some œdema were present. The hæmoglobin value of the capillary blood was 7·5 grammes *per centum* (50%), her blood group proved to be A, Rh-positive, and her blood gave a strong direct reaction with Coombs serum.

An exsanguination transfusion of 850 millilitres was given at the age of twelve hours, after which she manifested evidence of cardiac failure and received "Digoxin".

The jaundice persisted and deepened in intensity, and on her third day of life vomiting, rigidity of the limbs and tightly adducted thumbs were present. On her fourth day

a serum bilirubin estimation gave a result of 33 milligrammes of bilirubin per 100 millilitres of blood. Thirty-six hours after the onset of neurological signs a second exchange transfusion was given by the saphenous route; a total of 850 millilitres was administered.

The next day the jaundice had greatly decreased in intensity and the neurological signs had disappeared. The infant was discharged home, well, on the twenty-eighth day of life.

Observation in the subsequent six months has failed to reveal any evidence of permanent neurological damage.

CASE III.—C, a female child, was born on January 29, 1954, the second child of an Rh-negative mother and an Rh-positive father. Her elder sibling had been mildly jaundiced at birth, although her mother denied any previous miscarriage or transfusion. This infant was delivered normally at term, and her birth weight was seven pounds one ounce. Jaundice appeared before five hours and she was transferred to the Queen Victoria Memorial Hospital. She was jaundiced; hepatosplenomegaly and some bruising were present; her blood group was O(IV), Rh-positive, and her blood gave a strong direct reaction to the Coombs test; the hæmoglobin value of the peripheral blood was 12 grammes *per centum* (80%).

At eleven hours an exsanguination transfusion of 800 millilitres was given; at the end of the transfusion blocking antibodies to a titre of 1 in 64 were present in the infant's blood. The jaundice persisted and the urine was bile-stained. By the fifth day the jaundice had greatly increased in intensity and a petechial rash was present on the trunk; the infant's head was retracted, the thumbs were tightly adducted, and vomiting was a troublesome feature. Twenty hours after the appearance of neurological signs a second exchange transfusion was administered by the saphenous route, 800 millilitres of blood being exchanged.

The next day improvement was remarkable and the jaundice was fading. Two further simple transfusions were necessary for anæmia, and the infant was discharged home, well, on the fifty-third day of life. Observation during the subsequent ten months of life has failed to reveal any evidence of permanent neurological damage.

CASE IV.—D, a female infant, was born on April 17, 1954, at thirty-seven weeks' gestation, being delivered normally at the Queen Victoria Memorial Hospital; her weight was six pounds two ounces. She was the fifth child of an Rh-positive father and an Rh-negative mother, who had exhibited Rh-antibodies to a titre of 1 in 64 in this and previous pregnancies. The mother's second and third children had been jaundiced at birth, and her fourth infant had suffered from hæmolytic anæmia, which was treated with simple transfusions and ACTH. This infant was jaundiced almost at birth and exhibited hepatosplenomegaly; the hæmoglobin value of the cord blood was 12 grammes *per centum* (80%), the infant's blood group was O(IV), Rh-positive, and she gave a strong direct reaction to Coombs serum.

An exsanguination transfusion of 700 millilitres was given at sixteen hours after birth, at the end of which the hæmoglobin value was 15·9 grammes *per centum* (106%). No Rh antibodies remained in her blood, but the jaundice persisted and deepened. On the fourth day of life she had 28·5 milligrammes of bilirubin in 100 millilitres of serum. On the fifth day of life she was deeply jaundiced and lethargic, and exhibited persistent mouthing movements.

Twelve hours after the onset of signs of neurological involvement a second exsanguination transfusion of 700 millilitres was given, at the beginning of which circulating Rh antibodies were discovered to a titre of 1 in 2; these were presumably unstuck from the tissues and washed into the circulation in the five days intervening between transfusions.

The next day the jaundice had faded greatly, the hæmoglobin value was 13·5 grammes *per centum* (90%), and all neurological signs had disappeared. The infant had a simple transfusion for anæmia on the eighteenth day of life, and was discharged home, well, on the twenty-seventh day.

Observation in the subsequent seven months has failed to disclose any evidence of permanent neurological damage.

Conclusions.

In any case in which the jaundice deepens or persists after the initial exsanguination transfusion, a serum bilirubin estimation is of value in assessing the severity of the affection. If there are more than 20 milligrammes of bilirubin per 100 millilitres of blood, then it might be advisable to give a second exsanguination transfusion. The infant should be closely observed for the presence of early signs of kernicterus, such as drowsiness, vomiting, mouthing and later rigidity of the limbs and head retraction; but even after these signs have been observed, probably in a few cases for as long as forty-eight hours, it is possible to reverse the damage in the brain and return the infant to clinical normality by means of a second exsanguination transfusion.

From these observations it is impossible to determine whether kernicterus is due to the direct toxic action of bilirubin on the brain cells; but it would seem that there is probably some preexisting cell damage, wrought by anoxia or an antigen-antibody reaction or both, and that the hyperbilirubinæmia, if allowed to persist, will definitely establish permanent brain damage with the consequent disastrous clinical condition associated with kernicterus.

Summary.

The condition of kernicterus is defined. The ætiology would appear to depend on the following three factors: (i) A faculty, found in approximately 10% of Rh-positive babies, of producing Rh receptors capable of absorbing antibody. This faculty may be genetically inherited from the parents. (ii) Susceptibility of certain areas of the brain of the neonate to damage, which appears to be reversible in the early stages; but if hyperbilirubinæmia is allowed to persist, and pigmentation of the areas with bilirubin becomes established, the clinical manifestations of kernicterus seem inevitable. (iii) The presence in the brain of the neonate of a bilirubin-retaining lipid, which has a selective attraction for bilirubin of the indirectly reacting type, the conversion of this type to the directly reacting bilirubin depending on the integrity of the liver.

Four cases are presented, in all of which the infants exhibited the early manifestations of clinical kernicterus, in spite of early treatment with exsanguination transfusion; all of them appear to have completely recovered after a second exsanguination transfusion.

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Reviews.

Manual of Clinical Mycology. By Norman F. Conant, Ph.D., David Tillerson Smith, M.D., Roger Denio Baker, M.D., Jasper Lamar Callaway, M.D., and Donald Stover Martin, M.D.; Second Edition; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 8" × 5½", pp. 468, with 202 illustrations. Price: £3 1s. 9d.

THE first edition of this book was published in 1944. That the work has proved of practical value is shown by the appearance after ten years of the second edition with 88 new illustrations, making a total of 202. In the preface to the first edition it was pointed out that the classification of the pathogenic fungi had been simplified and the synonymics reduced, that for instance of *Candida albicans*, the fungus of the thrush, from 172 to 11. No wonder the medical mycologist got lost at times in the multitude of names for one specific entity! The preface to the second edition emphasizes that fungous infections are relatively, if not actually, more frequent since the new antibiotics have been controlling so many bacterial infections. Certainly the pathogenic fungi have been recorded more often in this journal during the last few years than was the case a decade back. It is disturbing to read that "50 to 75 per cent of the residents in the central part of the United States" have primary benign histoplasmosis infections, and it is not very

consoling to be told that "probably less than 0·1 per cent of primary infections . . . develop into the progressive and usually fatal form of the disease".

The book begins with actinomycosis, giving its definition, geographic distribution, source of infection, symptomatology, mycology, pathology, immunology, differential diagnosis and treatment and references to the literature. Then follow similar chapters on nocardiosis, the blastomycoses, coccidioidomycosis, histoplasmosis, cryptococcosis, candidiasis, infections with Aspergillus, Mucor and Penicillium, sporotrichosis, Madura foot, Rhinosporidium, the chromoblastomycoses, and the various dermatomycoses. All these diseases and their causative fungi are illustrated with many excellent photographs, often more than one to each "figure". The chapter on fundamentals of elementary mycology should help those not specially conversant with the terms used in the study of the fungi. Contaminants form a concluding chapter. An appendix of 21 pages describes mycological, pathological, immunological and dermatological methods, the last-named being followed by a formulary comprising wet dressings, dusting powders, ointments, paints and lotions.

It will be seen from the above summary of its contents that this "Manual of Clinical Mycology" covers as wide a field as it possibly can—and so it should with such a battery of authors. It should find a useful place in the clinical laboratory, should be helpful to the dermatologist who has to deal with so many diseases due to fungi, and may guide the worried general practitioner, the physician and the surgeon to a likely diagnosis in some of the rarer mycotic conditions.

History of the Second World War: United Kingdom Medical Series. Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.A., M.D., F.R.C.P., F.R.C.S. "The Royal Naval Medical Service", Volume I, Administration, by J. L. S. Coulter, D.S.C., R.N.; 1954. London: Her Majesty's Stationery Office. 9½" x 6½", pp. 528, with 13 illustrations. Price: 50s.

THIS volume of the "History of the Second World War: United Kingdom Medical Series" maintains well the high standard of its predecessors. Its scope is wide, including problems of recruiting, personnel, medical equipment, training, blood transfusion, preventive medicine, and research. The Royal Navy was depleted of medical officers, as were all the Services, by the rigid economy enforced after the first World War, and passed through the alarming period of shortage and slow recovery which has been detailed in the volume on Army administration. There was throughout the War an urgent and recurring necessity for the Royal Navy to put forcibly to the Medical Personnel Priority Committee its case for increased reinforcements of medical officers, but despite its efforts, although the number of medical officers increased sixfold, the proportion of doctors in the Naval Service actually diminished. Recruiting problems and particularly difficulties in regard to medical standards for recruits are discussed, and an interesting chapter records the experiments in neuro-psychiatry on H.M.S. *Standard* where a number of men of such low morale or of such a degree of mental instability as to make them unemployable as combatants, were segregated for physical and psychological treatment by a specially selected staff. The results amply justified the experiment.

The story of the emergence of the Royal Navy Blood Transfusion Service from a situation of almost complete dependence on other agencies, through the formation of Naval Bleeding Units, to a fully equipped and staffed organization adequate to supply all Royal Naval requirements of blood and serum, is a tribute to the cooperation by the Medical Research Council. The devoted service of the 113,918 donors in Yorkshire who contributed their blood to sailors during the War is gratefully acknowledged. How efficient the organization had become was shown by the supreme test of the Normandy landing, where the supplies of blood and serum for Naval units and the arrangements for its distribution were more than adequate. The Royal Naval Blood Transfusion Service made a valuable contribution to the Transfusion Services of the nation generally, and amply repaid the blood the Navy received from them in earlier days. The high standard of nursing and excellent morale (especially as prisoners of war) of the members of Queen Alexandra's Royal Naval Nursing Service are noted with appreciation. There were only two casualties from enemy action amongst members of this Service although many of its members were serving on torpedoed vessels. The greatest cause of wastage was marriage, and the fact that 327 sisters married and retired is quoted as "a tribute to the service as a whole". Approximately 100,000 women served in the Women's Royal Naval Service—25 of them women medical officers. A record of the use of 11 Naval Hospital Ships includes an account of the epic experiences of H.M.H.S. *Vita* which were surely sufficiently exciting and hazardous to satisfy the most adventurous. The development of the Naval Air Arm through the intermediate stage of the Fleet Air Arm of the Royal Air Force is described, with some reference to the associated medical problems. The longest chapter in the book is devoted to preventive medicine, in which the conclusion is reached that "the most important medical lesson learned in the War was that executive and combatant officers must be taught that the enforcing of hygiene measures to preserve the health, morale, and fighting efficiency of their troops is as important as any other military duty". This lesson was learnt by sad experience when Navy personnel, protected hitherto by the comparative immunity of ship life, were transferred in larger numbers than ever before to "the unhygienic hazards of a shore environment". The necessity to include general instruction in hygiene as part of the basic training of all personnel was soon realized and provided for, but the provision of firm executive support for the expert specialist advice given was not always immediately attained. While the medical personnel of the Royal Navy were throughout the War represented on Medical Research Council committees appointed to study clinical and other problems common to all the Services, there were many other problems peculiar to the Royal Navy for the solution of which the Naval Personnel Research Committee was appointed. The problems were many and diverse, and priority for consideration was often difficult to determine. The Navy fought in the hottest and the coldest regions of the earth, above, on, and below the sea surface, and each zone brought its own problems. Increasing complexity of armament and other equipment necessitated increased personnel for its service and at the same time encroached seriously on living accommodation. Of paramount importance thus became consideration of "habitability", which included general environment, temperature, humidity, overcrowding, diet and general amenities—all factors influencing standards of physical and mental health. The work of the Committee has already been outlined in the war volume on Medical Research, but it is here reported in greater detail. The volume concludes with an account of the work of the Dental Branch of the Royal Navy, who at times carried out their essential duties, especially when abroad, under conditions of extreme difficulty and inconvenience; however, they must surely have frequently worked under less congenial conditions than in the Jockeys' Hospital at Warwick Farm racecourse, Sydney, which is quoted as one of the strangest surroundings in which "pioneer work" was carried out. The valued assistance given by dental officers of the Royal Australian Navy and other Dominion Services is noted with appreciation. The authors have dealt very adequately with their task and present a very clear picture of the difficulties that confronted wartime Naval medical administrators and how their problems were solved.

Pomp and Pestilence: Infectious Disease. Its Origins and Conquest. By Ronald Hare, M.D.; 1954. London: Victor Gollancz, Limited. 8" x 5¼", pp. 224. Price: 12s. 6d.

THIS is a book that may be read with interest by the intelligent layman who wishes to acquaint himself with the way in which infective organisms that produce disease have acquired their properties of invasion, to appreciate the historical importance of the great pestilences that have literally decimated mankind, and to obtain some idea of the way in which, step by step, scientific control over most of them has been obtained. For these same reasons "Pomp and Pestilence" appeals to the medical reader who will find in its pages an epitome of the available records of epidemic and other parasitic diseases of the past.

The first chapter deals with parasites and parasitism. Parasitism may take three forms: symbiosis, where pest and parasite operate to their mutual advantage as in the association between white ants and the flagellates in their intestinal canal; commensalism (eating at the same table), illustrated by the hermit crab in a whelk shell carrying round with it a sea anemone; and pathogenesis, where the invasion by the parasite leads to disease. The hyadid cyst, more than "occasionally seen in Australia", takes pride of place as the largest parasite of man. But the author, quite rightly, of course, includes all the disease-producing bacteria and viruses as also parasites. He discusses how these organisms may have become parasitic and tells how the various kinds may pass from host to host, how the invasion may be overcome, how isolated communities may for long escape epidemics, and how the most dangerous parasites are in effect, by destroying their host, their own worst enemy.

Chapter two deals with man and his parasites. It is suggested that most of our infective diseases have been comparatively recently acquired and have not accompanied man

in his upward advance over a vast period of time. In this chapter are interesting references to the absence (with the exception of the smallpox outbreak in 1789) of epidemic diseases in our natives and the first recorded dates of scarlet fever (1843), measles (1851), diphtheria (1859), whooping-cough (1842) and influenza (1838) in Australia.

Medical men will be particularly interested in chapter three, "Parasites and Pestilence", dealing with epidemics of smallpox, plague, typhus, cholera and so on. Here the author shows his erudition in his many references to the classics, producing a very useful source of information. "Miasmas or Microbes", the title of chapter four, shows how the idea that disease was caused by gaseous emanations was gradually replaced by the proof that microbes were the cause. John Snow's and William Budd's researches make interesting reading to the medical man.

Then follow chapters on the reaction of the community, the reaction of the individual, and parasites and population. There are fourteen pages of notes and references.

We have enjoyed reading this book. Many of the references were new to us—that Titian had died of plague, that smallpox perhaps more than stout Cortés led to the conquest of Mexico. Alliteration probably brought "Pomp" into the title. Christmas is approaching. The price of "Pomp and Pestilence" is remarkably reasonable; it is a suitable gift for lay friends and for fellow practitioners who, as they read its pages, may well thank their stars, if we may still employ this mediæval phrase, that their lot was not cast in the Middle Ages when plague, pestilence and famine not infrequently decimated the population.

Spot Diagnosis: With Notes on Therapy. Compiled by the editors of *Medicine Illustrated*; 1954. London: Harvey and Blythe, Limited. Volume I. 9" × 6", pp. 128, with 110 illustrations. Price: 7s. 6d.

THIS book, presented by the editors of *Medicine Illustrated*, is divided into two parts. The first consists of 102 excellent photographs of patients or skiagrams illustrating different diseases arranged in a haphazard manner, and with each are given a few diagnostic clues, the correct diagnosis being printed on the following page.

The second part, headed "Notes on Therapy", consists of 15 pages devoted to brief reviews of recent therapeutic advances. Because of its brevity this section's usefulness is rather limited.

The authors state that they have endeavoured to include examples from all branches of medical practice, and that they have purposely avoided any system of sequence, so that no help to diagnosis is given by preceding pictures. Thus the book may be termed a pictorial "quiz", and students will find in it many types of cases that will confront them in their final clinical examinations. Tutors, too, could use this book to advantage when suitable patients are not available.

By the use of blocks previously presented in *Medicine Illustrated*, the price has been kept down to the very economical figure of 7s. 6d. in Great Britain, and because of this no student should miss having a copy of this very useful book in his possession.

Medical Mycology. Edited by R. D. G. Ph. Simons; 1954. Amsterdam: Elsevier Publishing Company. 9" × 6¼", pp. 460, with 342 illustrations, a few in colour. Price: 55s.

THIS book, representing the section of mycology of Volume II of "The Handbook of Tropical Dermatology and Medical Mycology", with the addition of a chapter on the development of medical mycology and a series of photographs, some in colour, of the cultural aspects to the most important fungi, will chiefly appeal to mycologist, dermatologist, and the physician interested in the systemic manifestations of fungous diseases. It should be a valuable reference work to students of the subjects.

The fungi are classified, and their physiology and techniques for identification are discussed in detail. A chapter on experimental infections in laboratory animals is included.

The medical aspects of the superficial and systemic mycoses are presented by the 36 authors in a concise, clear and comprehensive manner, and with a uniformity which does credit to the editor.

The controversial approach to moniliasis and onychomycosis is stimulating, though all who have had to treat these conditions will not be in complete agreement with such dogmatic statements as "Perleche is a manifestation of vitamin-B complex deficiency".

The illustrations are lavish, well chosen and of help in understanding the text. A complete index is provided. Chance checking proves this to be correct.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Legal Medicine: Pathology and Toxicology", by Thomas A. Gonzales, M.D., Morgan Vance, M.D., Milton Helpern, M.D., and Charles J. Umberger, Ph.D., with an introduction by Harrison S. Martland; Second edition; 1954. New York: Appleton-Century-Crofts, Incorporated. 10" × 7", pp. 1362, with 503 illustrations. Price: $22.00.

Based on extensive first-hand experience in the Office of the Chief Medical Examiner of the City of New York.

"The Digital Circulation", by Milton Mendlowitz, M.D., F.A.C.P.; 1954. New York: Grune and Stratton. 10" × 7", pp. 194, with 59 illustrations. Price: $6.75.

A comprehensive monograph covering the normal and the abnormal.

"Existence and Therapy: An Introduction to Phenomenological Psychology and Existential Analysis", by Ulrich Sonnemann, Ph.D.; 1954. New York: Grune and Stratton. 9" × 6", pp. 384. Price: $5.00.

An interpretation of current European developments in psychology.

"The Voice of Neurosis", by Paul J. Moses, M.D.; 1954. New York: Grune and Stratton. 8½" × 6", pp. 136, with four text figures. Price: $5.00.

Voice analysis from the points of view of the laryngologist and the psychiatrist.

"Clinical Psychiatry", by W. Mayer-Gross, M.D. (Heidelberg), F.R.C.P. (London), Eliot Slater, M.A., M.D. (Cambridge), F.R.C.P. (London), D.P.M., and Martin Roth, M.D. (London), M.R.C.P. (London); 1954. London: Cassell and Company, Limited. 10" × 7½", pp. 672, with 16 illustrations. Price: 50s.

The authors deal only with so-called "special psychiatry"; they have written for student, general practitioner, medical specialist and psychiatrist.

"A Pocket Medicine", by G. E. Beaumont, M.A., D.M. (Oxon.), F.R.C.P., D.P.H. (London); Third Edition; 1954. London: J. and A. Churchill, Limited. 7½" × 5", pp. 218. Price: 10s. 6d.

The book is for medical students when working in hospital and while waiting and travelling. It gives a short clinical account with appropriate treatment of diseases in the several systems.

"CIBA Foundation Symposium on Leuksemia Research", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 312, with 56 illustrations. Price: 30s.

This is the ninth of the "general volumes" in the Ciba Foundation Symposia. There are no less than 21 separate discussions on special subjects and one general discussion.

"An Introduction to Physical Methods of Treatment in Psychiatry", by William Sargant, M.A., M.B. (Cantab.), F.R.C.P., and Eliot Slater, M.A., M.D. (Cantab.), F.R.C.P., with a chapter on treatment of the epilepsies by Denis Hill, M.B. (London), F.R.C.P.; Third Edition; 1954. Edinburgh and London: E. and S. Livingstone, Limited. 9" × 6", pp. 370, with four illustrations. Price: 20s.

Intended primarily for the young clinician in psychiatry, for the general practitioner, and for the student.

"The Surgical Clinics of North America"; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Toronto: Nationwide Number. 9" × 6", pp. 334, with 76 illustrations. Price: £6 *per annum* with paper binding and £7 5s. per annum with cloth binding.

This volume consists of two parts. The first (nine articles and a foreword) is a symposium on thoracic surgery from the Faculty of Medicine, University of Toronto, Canada; the second (11 articles and a foreword) is a "nationwide" symposium on neurological surgery.

The Medical Journal of Australia

SATURDAY, NOVEMBER 27, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE ACTIVITIES OF THE CANCER INSTITUTE OF VICTORIA.

In the year 1936 the Argyll Government of Victoria brought down an Act to deal with the incorporation of an Anti-Cancer Council of Victoria. Sir Stanley Argyll, as well as being Premier of Victoria, was a practising radiologist, and was aware of the immensity of the cancer problem. The Act, which was known as the *Anti-Cancer Council Act*, 1936, stated that the objects of the Council were: (*a*) to coordinate in Victoria all activities in relation to research and investigations with respect to cancer and allied conditions and with respect to the causation, prevention and treatment thereof; (*b*) to promote and subsidize such research and investigations; (*c*) to provide maintenance and travelling expenses to persons in need who were suffering from cancer, to enable them to become inmates of or to attend a public general hospital or special cancer clinic for treatment; (*d*) to investigate the advisability of the establishment of special cancer clinics, and if thought advisable to establish such clinics; and (*e*) to facilitate the improvement of the treatment of persons suffering from cancer. The Lord Mayor of Melbourne became president of the Anti-Cancer Council, and an Appeals Committee set to work to raise money. A sum of some £60,000 was collected, an amount which even in those days could not be considered adequate for what was proposed to be done. The fund has since grown. The Anti-Cancer Council of Victoria, which, it should be pointed out, is affiliated with the British Empire Cancer Campaign, in its report and accounts for the year ended June 30, 1953, stated that the total funds of the Council at that date were £95,542. On June 30, 1954, at the opening of a clinic bearing his name, Sir Peter MacCallum, Emeritus Professor of Pathology in the University of Melbourne, described the formation of the Anti-Cancer Council of Victoria. He explained that members of the Council's committees surveyed existing conditions and tried to remedy some of the more obvious defects in provision for cancer treatment. After much discussion they drew up and adopted as a basis for action a statement of their ideas on organization for the best use of existing knowledge, leaving the part to be played in the wider issues to be dealt with as opportunity offered. They knew that surgery of high standard was widely undertaken in Victorian hospitals; but they found that conditions were otherwise in regard to radiotherapy. Radiotherapy machines, physicists, technicians and radiotherapists were expensive and were not available in sufficient numbers; there was not sufficient work for them to make wider distribution either economical or practicable. It was clear that a concentration of apparatus and personnel was necessary. In other words, an institution of a very special kind had to be created. It was at this time that New South Wales, being faced with similar problems, invited Dr. Ralston and Dr. Edith Paterson, of the Holt Institute, Manchester, to come to Australia, and Victoria and Tasmania were able to share in the benefit of their expert advice. Their advice, and the plans already made in Victoria, coincided so closely, and the Government was so impressed, that an allocation of money was made in the Victorian budget. A provisional committee had been on the point of adapting part of the Central or old Melbourne Hospital when the superior claims of the Queen Victoria Hospital to occupy that site made available to the cancer committees the building of the Queen Victoria Hospital. The Victorian Government appointed an interim committee under the chairmanship of Sir Peter MacCallum to report on the suitability of the Queen Victoria Hospital building for the establishment of an institute. The report was favourable, and with an officer of the Department of Health a recommendation regarding legislation was made to the Minister for Health; subsequently the Cancer Institute Board was established by Act of Parliament.

The Act, which was cited as the *Cancer Institute Act*, 1948, stated that an institute should be established which should have the following objects:

(*a*) to provide facilities for research and investigation with respect to the causation prevention diagnosis and treatment of cancer and allied conditions;

(*b*) to undertake such research and investigation;

(*c*) to provide in Victoria or elsewhere for the special training of persons in such research and investigation;

(*d*) to provide out-patient and in-patient hospital treatment at the Institute;

(*e*) to arrange for the provision at any hospital within the meaning of the *Hospitals and Charities Act* 1948 of special clinics at which patients may seek relief from conditions in respect of which appropriate treatment is available at the Institute;

(*f*) to provide hostels or make other arrangements where necessary for the accommodation of out-patients who are undergoing treatment at the Institute or at any clinic associated with the Institute;

(*g*) to provide at the Institute and, at any clinic established at any hospital, to arrange for the provision of—

(i) teaching facilities for medical students;

(ii) post-graduate instruction for medical practitioners; and

(iii) instruction to nurses technicians and physicists—

with regard to cancer and allied conditions, including the diagnosis and treatment thereof;

(*h*) to co-ordinate all activities arising from the foregoing objects.

The Act came into operation on March 29, 1949, and by the direction of the Honourable C. P. Garside, M.L.C., who was at that time Minister for Health, the inaugural meeting of the Board was held on April 27, 1949. It should be pointed out that the portion of the Cancer Institute which is concerned with all matters relating to the treatment of patients has been designated "The Peter MacCallum Clinic". This name is used in Launceston and Hobart, where radiotherapeutic clinics have been set up as the result of an agreement between the Governments of Victoria and Tasmania. An officer from the parent clinic visits these centres periodically. In connexion with the institute, useful work is carried out by an almoner's department. The institute contains a certain number of beds intended ultimately for intermediate and private patients; at present they are being used for public ward patients, but they cannot meet the demand which is made on the institute. When the next building alterations are completed in approximately fifteen months, the additions will be used for public ward patients. The almoner's department of the institute work in cooperation with the Austin Hospital, Heidelberg, and investigations are made so that certain patients can be admitted to the Austin Hospital. In a recent report of the proceedings of the Cancer Institute, it is stated that in certain circumstances where it is impossible under conditions existing at present in public hospitals to obtain the admission of a cancer patient who is severely ill, the senior almoner is authorized to utilize the services of some small private hospital. In certain circumstances also material assistance is given to patients from country centres who wish to find suitable board and residence while they are attending the institute for treatment as out-patients. One of the main services carried out by the institute, and one which is possibly not widely enough known, is the visiting nursing service. This service was established mainly for patients in poor circumstances who live within the metropolitan area. The plight of patients in the last stages of malignant disease for whom a suitable hospital bed cannot be found is grievous in the extreme. Any general practitioner who has such a patient may make application to the Cancer Institute, and the patient will be attended every day by a visiting nurse, whose entire time is devoted to work of that kind. The Cancer Institute has a large and representative board. Among its members are Professor Sir Peter MacCallum, who is the chairman of the Executive Committee of the Anti-Cancer Council of Victoria, and Dr. John P. O'Sullivan, who is chairman of the Medical and Scientific Committee of the Anti-Cancer Council. The present Medical Director is Dr. W. P. Holman, who recently succeeded Dr. R. Kaye Scott. Those who are interested in the treatment of cancer would be well advised to visit the clinic and learn at first hand of what it is doing and of what it hopes to do in the future.

The clinic has set out to provide adequate treatment facilities for lesions in all sites. To this end, X-ray therapy machines are installed or are being installed at the clinic to give beams of X rays from very "soft" qualities, produced at 20,000 volts, and used in superficial lesions, up to very penetrating beams of 4,000,000 volts, for use in treating deep-seated lesions. The equipment at present in use comprises a beryllium window X-ray tube unit operating between 20 and 60 kilovolts, two superficial therapy machines operating between 60 and 140 kilovolts, and nine machines operating at 250 kilovolts. The last-mentioned group of machines is at present used in the treatment of all deep-seated lesions, and will continue to be used for a large portion of such work; but for more adequate treatment, in some cases still more penetrating radiation is required, and preparations are now being made for the installation of a 4,000,000-volt X-ray machine known as a linear accelerator. This machine is being built in Britain, where five of them are being installed in various regional therapy centres. The accelerator is a development of the radar technique developed during the last war, and uses very high frequency radio waves to accelerate the electrons, which on hitting a target produce X rays. The accelerating tube is approximately six feet long, while the whole unit weighs about eight tons, and costs some £60,000. It will be housed in a room the concrete walls of which will be five feet thick. Observation of the patient by the operator will be made possible by installing in these walls a glass-sided tank filled with liquid.

Current Comment.

HOSPITAL EXPERIENCE AND MEDICAL REGISTRATION IN NEW SOUTH WALES.

In the New South Wales *Medical Practitioners Act* of 1938 a provision was included (subsection three of section 17) requiring a certain amount of experience before a registered person might practise his profession. The requirement was twelve months' service in a hospital or institution of a specified type or an equivalent form of training approved by the Medical Board. This provision was to have come into force on January 1, 1941. However, when the time came, wartime conditions made observance of the provision inexpedient, and its operation was suspended. After the war the number of new graduates from the University of Sydney Medical School each year was abnormally increased, and suitable hospital appointments could not be found for them all. To meet this situation, suspension of the hospital experience provision of the Act was continued.

At last, however, the situation seems to be under control. No further difficulty is expected, at least for the immediate future, in finding satisfactory hospital posts for new graduates. Elsewhere in this issue we publish a proclamation from the *New South Wales Government Gazette* of November 5, 1954, which brings subsection three of section 17 of the Act into effect for the first time. It will thus apply to successful candidates in this year's final examinations in medicine. For general information we have published the subsection (in its present amended form) in full. The provision is a wise one, and there will be general satisfaction that it has at last come into effect.

THE EFFECTS OF IMMOBILIZATION.

In a study published several years ago, J. E. Deitrick, G. D. Whedon and E. Shorr[1] described the effects of immobilization on a group of normal healthy men. These men were confined to bed with their pelvic girdles and legs in plaster casts for periods of from six to seven weeks. In discussing the undesirable effects that occurred or appeared likely to occur, Deitrick, Whedon and Shorr attached particular importance to urinary tract stone formation, impaired response of the circulation to the upright position, derangement in creatine metabolism, and loss of muscle mass and strength. These, it was considered,

[1] *Am. J. Med.*, January, 1948.

were likely to be real hazards for the patient recently immobilized for several weeks; they were perhaps matters of less concern for others, except those who were very ill or who were immobilized as the result of trauma, surgical procedures, poliomyelitis and similar disease conditions. For those in these last-mentioned groups, particularly if their immobilization (partial or complete) is for prolonged periods, the situation is not really clear, and not a great deal of light can be expected to be thrown on it by an experiment with healthy young men, to immobilize whom for a period likely to do real harm would be morally indefensible. The experiment mainly demonstrates that immobilization and bed rest for short periods offer a real hazard for certain types of patients.

Deitrick, Whedon and Shorr appear to have paid no particular attention to the age of the patient in their study, but a more recent report by Sverre Halvorsen[1] indicates that it is likely to be significant. Halvorsen lists the following physiological changes as being associated with immobilization: atrophy of muscles, increased protein catabolism, demineralization of bones with hypercalciuria and frequently occurrence of urolithiasis. He states that in adults the osteoporosis of disuse does not give rise to hypercalcæmia. In the growing organism, however, the bone atrophy may be greater and the hypercalciuria of larger degree, and hypercalcæmia with hypercalcæmic nephropathy may occur. This complication, he suggests, is probably not as uncommon as might be thought from the paucity of reports, which he briefly reviews. A case from his own experience was that of a previously active, nine-year-old boy with a slowly healing fracture of the left femur, who was immobilized for a period of twenty weeks. He developed severe polydipsia, polyuria, anorexia and vomiting, and his hearing became impaired. X-ray and laboratory investigations showed osteoporosis, nephrocalcinosis, renal insufficiency, hypercalciuria and hypercalcæmia. After mobilization, all symptoms and signs disappeared except the loss of hearing. The fracture healed in good condition.

Halvorsen discusses and rules out alternative diagnoses, the most important of which is hyperparathyroidism. Referring briefly to the relevant aspects of bone metabolism, he states that during growth of the skeleton bone is formed from cartilage in the zone of preparatory calcification. The deposition of calcium is influenced by nutritional and hormonal factors. Bone is constantly undergoing metaplasia. In some places new bone is formed, elsewhere bone is destroyed. The osteoblasts lay down osteoid tissue which is an organic extracellular substance rich in phosphatase and phosphorylase. These enzymes increase the concentration of phosphate ions and allow for the local deposition of calcium phosphate. At the same time, there are surfaces where bone is destroyed. The principal cells here are large foreign body cells called osteoclasts. The final result of the process is resorption of calcium from the bone to the extracellular fluids. The bone between formation and resorption is influenced by hormonal and nutritional factors and the degree of activity. It is postulated that mechanical stresses stimulate osteoblastic activity and thus the formation of bone. Conversely, during immobilization the osteoblasts lack stimuli, and formation of bone decreases; resorption of bone goes on undiminished, bone destruction exceeds formation, and decalcification occurs. It is further postulated, and this is particularly relevant to the case under discussion, that the speed of this process of bone atrophy will depend upon the degree of activity before immobilization. In a very active individual, the equilibrium will be more upset by immobilization than in one used to less activity. Consequently, greater calcium resorption and hypercalcæmia are likely. Halvorsen states that such a high degree of activity is found in children and adolescents, and hypercalcæmia following immobilization has so far been encountered in this group.

In the light of these theories, Halvorsen traces out the probable course of events in the case reported. He points out, however, that so many children of both sexes are immobilized for long periods and probably do not develop hypercalcæmia, that one must search for additional factors. Examination of the evidence in this and other cases indicates that a very strict degree of immobilization is probably essential for the development of hypercalcæmia. Very slight increases in activity are probably sufficient to adjust the calcium balance. Renal injury is very common in conditions associated with hypercalcæmia, and anorexia and vomiting are quite well known, but hearing loss is not generally recognized as a complication of this state, and particular interest attaches to its occurrence. Although Halvorsen is of the opinion that the hearing loss was due to hypercalcæmia, there is not a great deal of evidence, experimental or otherwise, to justify this view other than the course of events of the boy's illness. The boy and his parents are reported to have noticed decreased hearing acuity when his general condition was at its worst. This was definitely later than the date of the operation, following which the boy received seven grammes of streptomycin. Halvorsen quotes the five cases of deafness reported by J. A. Simpson[2] as occurring in association with the hypercalcæmia of hyperparathyroidism. No further comment seems justified at present.

Drawing some practical conclusions from his own experience in this case and from that of others, Halvorsen points out that osteoporosis, hypercalcæmia and nephropathy may occur in previously active children who are suddenly and very strictly immobilized. Additional factors, such as slow fracture healing, renal disease and a diet of high calcium content, will facilitate the development of these complications. The condition is reversible if diagnosed and treated early, otherwise serious complications may persist. If diagnostic differentiation from hyperparathyroidism is difficult, one should probably try a short period of mobilization before exploratory operations are decided on. The treatment consists of mobilization, forcing of fluid intake and a diet of low calcium content. The condition can probably be prevented with physical therapy and early mobilization, and it may be controlled by testing the specific gravity of the morning urine throughout the immobilization period and by determining the serum calcium content in suspected cases.

[1] *Acta med. scandinav.*, Volume 149, Fascicle 6.

THE PATHOGENESIS OF ACUTE PANCREATITIS.

THE term acute pancreatitis has been undergoing critical evaluation in recent years. In the past it was thought of essentially in terms of acute catastrophic inflammation of the pancreas with associated necrosis and hæmorrhage. However, as H. L. Bockus, J. L. A. Roth and R. I. Sifre have pointed out,[2] more recent correlation of pancreatic serum enzyme determinations with operative or necropsy observations has permitted the recognition of less severe instances of acute pancreatitis with little or no necrosis. This milder grade of inflammation of the pancreas has been designated acute pancreatic œdema (or interstitial pancreatitis) and has been associated usually with milder symptoms and signs, lower serum enzyme values and less likelihood of the occurrence of delayed hypocalcæmia. Bockus, Roth and Sifre go on to state that acute œdema of the pancreas has been recognized most often in association with acute episodes of biliary tract disease, but it should be borne in mind that any mechanism capable of causing acute necrosis may at times cause only œdema without concomitant necrosis of the pancreas. They suggest that the only tenable concept concerning the pathogenesis of acute pancreatitis is that all grades of acute inflammation of the pancreas, with or without massive necrosis, are but manifestations of the same basic process representing varying degrees of inflammatory response to the insulting agent. They list the following factors as entering into the degree of the response: (a) the *modus operandi* of the inciting cause or causes, (b) the presence or absence of obstruction to the outward flow of pancreatic juice, (c) the presence or absence of antecedent pancreatic inflammation, (d) the degree of escape of pancreatic enzyme into the surrounding tissues, and (e) the

[1] *Brit. M. J.*, February 27, 1954.

[2] *Surg. Clin. North America*, December, 1951.

presence of absence of a disturbance in the blood supply to the pancreas.

A number of these factors have been reexamined constructively by M. A. Block, K. G. Wakim and A. H. Baggenstoss in a series of experimental studies on rats.[1] Their experiments were particularly concerned with the effects of obstruction of the pancreatic duct, of the production of ischæmia of the pancreas and of stimulation of secretion of the pancreatic juice. They found that after partial or complete obstruction of the flow of pancreatic juice, pancreatic changes occurred consisting of œdema, inflammation, degeneration and atrophy of acini, and interpancreatic and extrapancreatic fat necrosis. Significant pancreatic parenchymal necrosis did not occur. Occlusion of the common bile duct so as to permit the mixing of bile and pancreatic juices under physiological pressures did not alter the changes produced as compared with those occurring after obstruction of the flow of pancreatic juice without admixture with bile. The production of temporary or permanent ischæmia in addition to obstruction of the pancreatic ducts resulted in pancreatic parenchymal necrosis varying in severity with the degree of arterial obstruction to the pancreas. Complete devascularization of large portions of the pancreas generally resulted in the appearance of ischæmic infarcts, but occasionally acute pancreatitis occurred with pancreatic necrosis and with fat necrosis without dilatation of ducts and acini.

The data obtained in this study are regarded by Block, Wakim and Baggenstoss as supporting in general the concepts derived by other investigators on the pathogenesis of acute pancreatitis. It appears that acute obstruction of the pancreatic ducts will result in at least one form of acute pancreatitis with peripancreatic fat necrosis, but parenchymal necrosis is generally absent. Degenerative changes and atrophy of the acinar cells occur, but necrosis usually does not. The production of a common channel between the bile and pancreatic ducts does not appear to modify the changes significantly. If ischæmia is added to obstruction of the pancreatic duct, parenchymal necrosis is produced in addition to other pancreatic damage. Hæmorrhage accompanies the necrosis in some instances, especially if the ischæmia is temporary. The investigation further showed that in the rat acute pancreatitis with fat necrosis and parenchymal necrosis could be produced by severe and extensive pancreatic ischæmia alone. Generally, however, these lesions had the appearance of focal ischæmic infarcts and did not duplicate the histological appearance of diffuse pancreatitis observed in man. Dilatation of the ducts and acini was absent. Evidently, as other workers have noted, fat necrosis can result independently from either severe pancreatic ischæmia or obstruction of the pancreatic duct.

On the question of the relationship of vascular disease to acute hæmorrhagic pancreatitis, Block, Wakim and Baggenstoss refer to the absence from the literature of convincing evidence that vascular factors are important in the initiation of this condition. Nevertheless they consider that they may be of great importance in the progression and ultimate outcome of the disease process. Thus, whether obstruction of the pancreatic duct in man results in a relatively benign form of acute œdematous or interstitial pancreatitis, or whether it terminates in diffuse pancreatic necrosis may depend on vascular factors. The results of the present experiments lend support to this viewpoint.

The investigators were unable to demonstrate definitely that stimulation of the secretion of pancreatic juice in rats exaggerated the changes in the pancreas after obstruction of the pancreatic ducts, and certainly significant pancreatic necrosis did not occur under these conditions. It has been suggested that after injury to the pancreas a secretory inhibition of the pancreas occurs which resists all stimuli. However, Block, Wakim and Baggenstoss consider it obvious that the pancreas must be secreting simultaneously in the presence of obstruction of the duct before dilatation of the duct and the changes due to the action of the constituents of the juice can occur. It has been established that the enzymes of pancreatic juice may be activated in the pancreatic ducts without contact with the intestinal juices, but activation apparently is more pronounced if the latter occurs. Evidently the fat necrosis and other changes observed in this study appear when extravasation of the pancreatic enzymes into the œdema fluid occurs after obstruction of the duct, or as a result of rupture or disintegration of ducts.

Block, Wakim and Baggenstoss are rightly hesitant in the correlation of their experimental results with clinical findings in man. Nevertheless, they point out that it may be significant that the dilatation of ducts and acini and the degenerative changes and atrophy of acinar cells, which occur after obstruction of the pancreatic duct alone, simulate the histological changes observed in fibrocystic disease of the pancreas in children, which has been recently attributed to congenital atresia of the pancreatic ducts. Associated with these changes in the pancreas of the rat, there is tremendous interlobar œdema, and numerous mononuclear leucocytes and occasional polymorphonuclear leucocytes are present. This exudative reaction is not observed in fibrocystic disease, but approaches that observed in so-called acute œdematous pancreatitis or acute interstitial pancreatitis, which frequently is encountered by surgeons during the course of operations on the biliary tract; it is, indeed, much more common than has been realized in the past, being commoner than the better known acute hæmorrhagic pancreatitis. Thus, it is conceivable that acute œdematous pancreatitis is due to obstruction of the pancreatic duct. When pancreatic necrosis or hæmorrhagic pancreatitis occurs, however, ischæmia may be the additional factor which is responsible for these changes. To produce extensive and spreading acute pancreatic necrosis, a large area of pancreas evidently must be separated from the arterial blood supply. In relation to another entity, it is pointed out that with permanent occlusion of the pancreatic ducts, fibrosis of the pancreas occurs, and it is thus conceivable that repeated acute obstruction of the pancreatic duct may be a factor in the pathogenesis of the clinical entity known as chronic relapsing pancreatitis.

If the factors of obstruction of the pancreatic duct and ischæmia are accepted as of basic importance in the causation of pancreatitis, one may then look for clinical conditions inducing these. Obstructive factors will include spasm of the ampullary region, stones in the common or pancreatic duct, œdema of the papilla or duodenal mucosa, and stricture or cicatricial stenosis of the papilla. To be certain of the cause of ischæmia when it is present in acute pancreatitis is difficult, as Block, Wakim and Baggenstoss point out, but œdema and inflammation, thrombosis of vessels, the action of enzymes on vessels resulting in their occlusion and vascular spasm all may be important factors.

[1] *Surg., Gynec. & Obst.*, July, 1954.

RETROLENTAL FIBROPLASIA.

THE July, 1954, issue of the *British Journal of Ophthalmology* is given over to reports on the study of retrolental fibroplasia by Norman Ashton *et alii*. Ashton's work on retrolental fibroplasia and on the retinal circulation is now widely known. In the first paper he describes in detail the pathological findings in eyes showing early stages of retrolental fibroplasia, and he is able to simplify the understanding of the course and development of the disease by dividing it into three pathological stages. Stage I is characterized by proliferation of the vaso-formative tissue of endothelial and mesenchymal cells in the inner layers of the retina with early eruption of the tissue through the internal limiting membrane into the vitreous. In Stage II there occur commencing retinal detachment, extension of fibrovascular tissue into the vitreous and the function of fibrous strands, and in Stage III total retinal detachment and formation of dense retrolental fibrous membrane occur, followed by late atrophic changes. Ashton's findings agree in the main with those of other workers, but differ in that he finds that the region of the ora is normal and the *pars ciliaris retinæ*, not firmly attached in premature eyes, is readily detached if there is detachment of the *pars optica*. In 280 cases of retrolental fibroplasia studied, oxygen had been administered in every

instance. Ashton has never seen the disease in stillborn or short-lived premature infants nor in fully mature infants. The second paper in collaboration with B. Ward and G. Serpell, and the third paper with Charles Cook, deal with the effect of oxygen on the developing retinal vessels. Full-term kittens were used in the study of oxygen concentration on developing retinal vessels, since it was found that the degree of retinal vascularization in the full-term kittens at birth and in the ensuing three weeks is comparable with that in the premature baby. It was discovered that high concentrations of oxygen exert an obliterative effect on the ingrowing retinal complexes which only partially reopened when the animal was transferred to air. In the final paper by Ashton and Charles Cook the effect of oxygen on the retinal vessels is observed directly through the limbal window. These papers demonstrate the meticulous care, attention to detail and ingenuity of the workers concerned. Their experiments leave no doubt as to the nature and cause of retrolental fibroplasia.

RADICAL CANCER SURGERY.

WHEN the comparatively recent history of cancer treatment is reviewed, it is interesting to note the changes in technique for eradication of this scourge. The surgeon, aided by modern anæsthetics and his team of resuscitation experts together with the antibiotics, is enabled to perform initial operations of greater magnitude than ever before. This is a mixed blessing and a source of misgiving to many. In spite of performing extensive primary resections of stomach or bowel, O. H. Wangensteen *et alii*[1] now publish an interim report on their experiences in the use of the so-called routine second (or even sixth) look explorations of the abdomen to find if there is any residual cancer or recurrent cancer present after the initial extensive operation for removal of the affected abdominal organ. These abdominal explorations are repeated at intervals of four to six months until no more cancer is found. The rationale is that one cannot always remove all cancer at the original operation because one cannot always tell how far it has spread. The second look six months later may reveal the increased growth of the previously hidden cancer. The cases reported are sufficiently interesting to make one keep an open mind on whether these operations are worth the inconvenience, if not danger, to the patient of having a laparotomy each six months, especially as the extraordinary differences of growth in carcinoma in different individuals are well known. Wangensteen and his colleagues admit that the "second look" operation is still an experimental venture, but it is well worth watching this surgical procedure until something better offers.

A. Brunschwig and W. Daniel[1] give an account of their experiences with radical cancer surgery of the pelvic organs over the past seven years. Their operation of total exenteration of the pelvis means excision of the bladder, lower parts of the ureters, vagina, uterus and adnexæ, rectum and lower part of the sigmoid colon, pelvic lymph nodes and pelvic peritoneum. The ureters are implanted into the upper part of the sigmoid colon, a wet colostomy resulting. In anterior exenteration, the bowel is left intact and the ureters are implanted into the sigmoid colon. The indications for these operations are widespread cancer involvement of the pelvic organs, often after failure of radiotherapy. Brunschwig and Daniel find that these operations are still received with scepticism in many quarters and agree that they are palliative only. While their total mortality rate is 26·6%, the five-year survival rate (they describe it as a cure rate) was 12% among 104 patients. This is a development of cancer surgery that will be watched with interest. However, before one accepted a judgement on its worth from figures and statistics, it would be necessary to examine the surviving patients in person to see what their life was like and if their ordeal had been worth while.

So far there have been no rival methods to challenge the surgeon in the abdomen, but a different story is present in the development of the treatment of breast cancer. On the one hand, we have the advocates of surgery more extensive than Halsted's radical mastectomy, with the surgeon exploring inside the thorax and up to the supraclavicular region. On the other hand, Professor Robert McWhirter, of Edinburgh, using his method of simple mastectomy only *plus* radiotherapy, has produced better survival rate figures than anyone using radical surgery. As in all cancer surgery, the vagaries and different speeds of growth, infiltration and formation of metastases in what histologically seems the same cancer may vitiate the results of even the most carefully controlled series; so much is this so that competent investigators such as W. W. Park and J. C. Lees[1] doubt whether the five-year survival rate is affected by treatment at all—a statement which has been rapidly brushed aside (but not disproved) by surgeons of all countries. Be that as it may, the practising surgeon, using whatever operative technique he believes to be the best, must teach his students and himself the diagnosis of cancer of any organ at the earliest possible moment. No longer should we wait for the obvious breast carcinoma of the older text-books. Any doubtful swelling in the breast, no matter how small or recent, should be excised and examined as soon as possible. In this way lies the only real hope of success in the treatment of cancer rather than in the use of exenterations or "second looks", which after all are only palliative procedures for late cancers.

[1] *Surg., Gynec. & Obst.*, September, 1954.

PEYRONIE'S DISEASE.

THE condition of chronic indurative cavernositis derives its commonly used name of Peyronie's disease from the French surgeon François de la Peyronie, who described it over two hundred years ago. It is characterized by the development of indurated plaques in the *corpora cavernosa* and results in curvature of the penis with pain during erection and difficulty of sexual intercourse. Of unknown ætiology, it has long presented a very disturbing problem to urologists. No treatment as yet recommended has been outstandingly successful. A new treatment recently suggested by G. H. Teasley[2] consists of direct injection of a solution of cortisone into the fibrous plaque. It is known that cortisone can delay the development of fibroplasia, and that it has the capacity of inhibiting the genesis of fibrils, as demonstrated in cultures of fibroblasts. The question of using this drug locally in Peyronie's disease was introduced after a general surgical associate had shown Teasley the results of injection for adhesions around a joint. The first patient, aged sixty-five years, had used tocopherols for several months without any success and had been unable to have intercourse. It was explained to him that this was an entirely new idea, but he consented to the experiment, and 25 milligrammes of cortisone in 1·5 millilitres of normal saline solution were injected directly into a dorsal fibrous plaque. The injections were repeated weekly to a total of six. Before the end of that course he achieved practically normal erection and intercourse, and when interviewed at intervals up to eight months later, he reported no recurrence. Teasley had experience of the treatment in three other cases with results as good, or almost as good, as in the first case. Reports of the effect of the same type of treatment concerning another 20 cases were received from five other urologists, 12 being from W. C. Baum, of the University of Michigan, who reported moderate results in most cases, and good results in a few. The technique involves infiltration of the skin and subcutaneous tissues down to and around the plaque to control the pain of injection, no general anæsthetic being used. It is essential to use a strong Luer-Lok syringe with an 18 to 20 gauge needle. Unless this type of syringe is used, the needle will be blown off the syringe, since the point of the needle must enter the dense plaque and the pressure necessary to secure injection is great. Twenty-five milligrammes of cortisone are dissolved in 1·5 millilitres of normal saline, and this contains 0·25% of procaine to reduce pain. The injection is easier if the syringe has metal finger rings.

[1] *Ibidem*, August, 1951.

[2] *J. Urol.*, May, 1954.

Abstracts from Medical Literature.

DERMATOLOGY.

Treatment of Molluscum Sebaceum.

R. FINNEY (*Lancet*, April 24, 1954) states that the condition known alternatively as *molluscum sebaceum*, *molluscum pseudocarcinomatosum* or kerato-acanthoma is now established as an entity. The treatment is not generally agreed on. Indeed, *molluscum sebaceum* is a self-limiting, self-healing condition, and it might be argued that any treatment was therefore unnecessary. Against this, its natural course may last up to six months; hence treatment would appear to be justifiable to procure early healing. The danger of treating a squamous epithelioma as *molluscum sebaceum* has been emphasized. This is more likely in the early stage before the formation of the central keratin plug. There appear to be two main methods of treating this condition—surgical removal and radiotherapy. Surgical procedures have included diathermy or electrolysis, local incision and suture, and excision or curettage of the superficial parts of the tumour. The author felt that treatment by radiotherapy alone could play a part in the treatment of the condition, just as for other benign conditions, such as hæmangiomata, keloid scars and plantar warts. To determine the optimum dose to cause regression of the tumour, 25 subjects were treated by radiotherapy alone. A Chaoul tube was used at a voltage of 60 kilovolts, four milliampères, and a half-value layer of 3·3 millimetres of aluminium. A single exposure was given. Four separate dosage levels were used. Group A had 2250*r* to 2500*r*, group B had 1750*r* to 2000*r*, group C had 1500*r*, and group D had 1200*r* to 1250*r*. The average tumour-regression time for all groups was about four and a half weeks. The author considers that *molluscum sebaceum* may be effectively treated by radiotherapy. A satisfactory tumour-regression rate follows a dosage level of 1500*r* to 1750*r*. A dosage above this produces an unnecessarily severe and prolonged reaction.

Dysidrosis (Pompholyx).

W. S. SHELLEY (*Arch. Dermat. & Syph.*, September, 1953) states that, for the purpose of his report, dysidrosis may be defined as an acute recurrent, non-inflammatory vesicular eruption strictly limited to the palms and/or soles. The process is not seen elsewhere except as an extension on to the lateral surfaces of the fingers or toes. It occurs in both sexes and more commonly in adults. The primary lesion is a vesicle, which is clear, deep-seated and non-erythematous. It is sterile. The vesicles may be pruritic, and may enlarge and coalesce to form bullæ. The eruption is symmetrical. The course is one of sudden onset and rapid evolution followed by spontaneous involution. The vesicles do not rupture, and they exude serum spontaneously. Repeated attacks occur as a rule, seasonal exacerbations being common in summer. Hyperidrosis of the palms and soles is the usual concomitant of dysidrosis. The lesions may become infected; or, owing to topical medication, contact dermatitis and secondary eczematization may occur. Histologically, dysidrosis manifests itself as a superficial vesicle in the epidermis. Most recent studies prove the vesicle to be independent of the sweat duct. The author considers that dysidrosis is a non-specific reaction pattern of palmar and plantar skin. The cause is not known in the majority of cases. Hutchison and Fox noted the psychosomatic factor. Primary fungous infection with a dermatophyte is another cause of dysidrosis. Wittkower has reported that emotional factors were ætiologically operative in 40 or 50 cases of dysidrosis. Dysidrosis of the palms (and soles) can arise on the basis of an allergic state in patients presenting an active inflammatory fungous infection of the feet. Proper topical medication minimizes the opportunity for secondary infection and contact eczematization. Compresses are soothing in the early stages of the disease; later, an emollient cream is applied. If secondary infection has occurred, local or systemic antibiotic therapy may be appropriate. Penicillin in general is avoided in view of its capacity to induce dysidrosis. Sedatives may be employed to allay tension. Atropine may be of value, as well as "Prantal" and "Banthine" and ganglionic blocking agents. X-ray therapy has no proved value.

Dermatitis of the Hands.

R. L. SUTTON AND S. AYRES (*Arch. Dermat. & Syph.*, September, 1953) list the following ten mechanisms provoking inflammatory skin disease of the hands: (i) trauma of all kinds, (ii) contact injury by primary irritants, (iii) contact injury to sensitizers, provoking itching, redness and vesiculation when the skin is touched, even in minute concentration, (iv) mechanisms of injury by chemical substances absorbed and provocative of intoxication, of allergic reactions such as *dermatitis medicamentosa*, including fixed eruptions, or of food allergy, (v) the mechanism of locally invasive parasites, including viruses, bacteria and fungi as well as animals such as *Acarus scabiei*, (vi) the mechanism of focal infections, which influences the distant site, perhaps by supplying circulating parasites or by-products of parasites to be filtered out by an already existing inflammatory reaction, (vii) the mechanism of nervous and mental or psychiatric abnormality, including anxiety, excoriation and self-mutilation, (viii) the mechanism of cutaneous changes due to nutritional deficiencies, metabolic defects and hormonal imbalances, (ix) lowered resistance, (x) combinations of these mechanisms. The authors discuss the ætiological classification of dermatitis of the hands under the following headings: (i) physical agents, (ii) chemical agents, (iii) "id" reactions, (iv) dermatitis of internal origin, (v) food allergy, (vi) nutrition, (vii) parasitism, (viii) infections from the interior, comprising focal infections, (ix) dermatitis of unknown ætiology, (x) pompholyx or dysidrosis. On the question of management the authors state that dermatosis is likely to respond succesfully if the physician achieves at one time elimination of contactants, of parasites, of focal infections, and of defects in nutrition and metabolism with especial reference to vitamins, proteins, liver function and hormonal balance. Use of the safest medicines one can select for topical application, avoidance of over-treatment and wisdom in the use of X-ray therapy are advised. The authors state that vesiculo-pustular dermatitis of the hands is seldom fungous infection, but is usually bacterial infection which is generally made worse by fungicidal medication. Erosion of the *cervix uteri* is a common focus of infection pertinent to chronic dermatitis of the hands in women, and as such should be looked for in every case. Over-ingestion of caffeine has an importantly deleterious effect in promoting irritability of the skin, accumulation of fatigue and chronicity of dermatitis.

White Nails in Hepatic Cirrhosis.

R. TERRY (*Lancet*, April 10, 1954) discusses opacity of the nailbed causing apparent whiteness of the finger nails and its occurrence in cirrhosis of the liver and some other conditions. He states that fully developed white nails exhibit a groundglass-like opacity of almost the entire nailbed. It extends from the base of the nail, where the lunula is indistinguishable, to within one or two millimetres of the distal border of the nailbed, leaving a distal zone of normal pink. The condition is bilaterally symmetrical. The opacity of the nail is demonstrated by squeezing the blood into the terminal phalanx. The normal nailbed becomes congested, whereas any whiteness of the nailbed is unaffected by congestion, and indeed is accentuated by contrast with the congested pink zone. White nails were found in 82 of 100 consecutive patients with cirrhosis. The ætiology of the cirrhosis was alcoholic in nine, post-necrotic in seven, and cholangiolitic in two. In nearly all cases in which white nails were present, all the nails in both hands were affected. Severe longitudinal ridging of the nails was common, as was some thickening. Of patients with spider nævi, 37% had severe or gross white nails, compared with 22% of those without nævi. The white opacity is entirely different from leuconychia, for in the latter condition the white areas are in the nail itself, and as the nail grows they steadily move towards the finger tip; in white opacity described by the author neither the white opacity nor the distal pink zone alters as the nail grows. The nail itself is normally transparent, and the opacity evidently results from some change in the nailbed. It is suggested that white nails are endocrine stigmata comparable with spider nævi, palmar erythema, cutaneous striæ and gynæcomastia. Well-developed white nails are apparently rare in healthy adults and their detection therefore may be of some importance.

Depigmentation Properties of Monobenzylether of Hydroquinone.

L. FORMAN (*Brit. J. Dermat.*, November, 1953) states that in 1940 Schwartz, Oliver and Warren described cases of occupational leucoderma occurring in a tannery. The offending substance was an antioxidant, monobenzylether of hydroquinone, which was commercially known as "Agerite Alba" and was added to the rubber mixture. Denton *et alii* (1952) described the results of patch tests with 10% and 30% monobenzylether of hydroquinone applied continuously for thirty days, on five coloured and two white patients. Depigmentation developed in two of five Negro subjects, the higher concentration of hydroquinone giving

greater depigmentation. With similar concentrations of hydroquinone, another two of the five Negro subjects developed slight depigmentation and two developed dermatitis, one with a secondary increase of pigmentation. No change was noted in the skin of two white males. Denton recorded the result of treatment in a case of generalized lentigenes, with considerable improvement over the face. A patient with chloasma following pregnancy was treated with 30% ointment successfully with resulting depigmentation. Fitzpatrick believes that 5% "Agarite" in a water-soluble base could be applied two or three times a day and should be reasonably safe when used for one to four months. The patient with chloasma applied an ointment containing 5% monobenzylether of hydroquinone once a week for five months to the left side of the forehead and for three months to forehead and cheeks. No inflammatory reaction occurred, but depigmentation was definite. The author sums up as follows: (i) Monobenzylether of hydroquinone has a specific blocking action on the formation of skin pigment, perhaps at the dopa-melanin stage. (ii) The compound can also act as a skin sensitizer; the depigmentary and sensitizing properties do not appear to run parallel. (iii) On white skin, a dermatitis caused by this chemical may be followed by increase of pigmentation. (iv) A case of chloasma is reported, in which depigmentation followed the application of monobenzylether of hydroquinone without evidence of dermatitis.

UROLOGY.

Nephro-Ureterectomy and Heminephro-Ureterectomy in Childhood.

H. M. SPENCE (*J. Urol.*, February, 1954) discusses the indications for, and technique of, nephro-ureterectomy in infancy and childhood. He considers that this operation has not received the recognition and acceptance that it merits, and he hopes that publication of his experiences will help to this end. In 1921, Beer introduced his "aseptic nephro-ureterectomy". The essential features of this are as follows: (i) Lumbar nephrectomy is performed, the kidney being left attached to the ureter, which is freed as far down as possible. (ii) The wound is closed, the kidney and upper part of the ureter being left outside covered with a sterile towel, and dangling there. (iii) The patient is turned onto his back, and a mid-line suprapubic extraperitoneal approach is made to the lower part of the ureter; this is easily recognized by pulling on the upper part of the ureter to render the lower part taut. (iv) The ureter is stripped down to the bladder, where it is doubly ligated and divided. (v) The kidney and entire ureter are now withdrawn from the anterior end of the lumbar wound, without spilling of its contents. (vi) The lower wound is closed, with tube drainage. The indications for nephro-ureterectomy originally given by Beer are those followed by the author: (i) papillary tumour of the renal pelvis (and/or the ureter), (ii) renal tuberculosis, (iii) a destroyed kidney, with hydroureter, secondary to obstruction low down in the duct. Where the renal destruction affects only one portion of a reduplicated pelvic-calycine system, the destroyed part can easily be removed; and when its draining ureter is also dilated, the operation of heminephroureterectomy is carried out down to the point where the affected ureter either joins the other duct or opens separately into the bladder. It is preferable to ligate the blood vessel supplying the diseased segment before incising the kidney. Then the incision is made at the junction of firm and thinned-out parenchyma. When the renal vessels branch only after entering the kidney, temporary compression of the pedicle is applied. Hæmostasis is effected with horizontal mattress sutures of catgut. Complicated suturing techniques are not nearly so safe or effective. The remaining portion of the kidney is suspended in a high normal position. The temporary pressure on the pedicle is released as soon as the mattress parenchymal and final capsular sutures have been placed. The rest of the ureterectomy technique is concluded as described above. Beer originally reported 40 cases of one-stage nephro-ureterectomy without a death. The author reports seven cases with excellent results.

Cystectomy and Carcinoma of the Bladder.

E. SAYEGH (*J. Urol.*, June, 1954) reviews 106 cases of advanced carcinoma of the bladder (99 in males and seven in females), in order to try to determine which of three major surgical procedures offers the greatest measure of palliation. The procedures are (i) bilateral transplantation of the ureters only, (ii) ureteral transplantation and total cystectomy (this includes prostato-vesiculectomy in the male and panhysterectomy in the female), and (iii) radical cystectomy, which includes total cystectomy and complete clearing out of all pelvic glandular areas. The immediate postoperative mortality was about the same in all three groups, namely, 10%. In the first two groups, there were no survivals beyond the third year (21 and 23 patients respectively). In the third group of 62 patients in all, eight patients have survived more than three years. The author considers that radical cystectomy is the operation of choice in these advanced cases.

Histopathology in Prognosis of Renal Neoplasms.

W. S. BARRETT AND E. J. MCCAGUE (*J. Urol.*, June, 1954) have made an analysis of 199 well authenticated and histologically proved malignant neoplasms of the kidney met with in two large general hospitals in Pittsburgh, United States of America, over a period of twenty years. Of the patients affected, 102 were subjected to nephrectomy. Only 20 of these survived a five-year period after operation. Of these 20 survivors, 13 continued to be well for ten years or more; this represents only 6·5% of the original total of 199 cases. Nephrectomy with X-ray therapy offered a slightly better prognosis than nephrectomy alone. Study of the adenocarcinomata of the renal parenchyma (hypernephroma) indicated that the different cellular configurations were merely variants of the same tumour. The ones which had the greatest degree of cellular differentiation and cellular grouping were the slower growing tumours. The cells which were undifferentiated and anaplastic were the more rapidly growing and more malignant. In the group of transitional cell tumours of the renal pelvis there were three of the squamous type, all of which had extensive metastases, with death within one year. The authors state that the greatest weakness of current treatment in these conditions is the lateness of diagnosis. Unless the disease can be diagnosed in the early stage, before metastasis, even improved surgical treatment will effect very little change in the late results.

Carcinoma Complicating Vesical Diverticulum.

R. F. MAYER AND T. D. MOORE (*J. Urol.*, March, 1954) state that the occurrence of carcinoma in a vesical diverticulum was once looked on as a clinical rarity, but in recent years it has been recognized more often, and now about 150 cases have been reported. The authors describe eight additional cases. They state that the diagnosis is difficult, and in the reported cases recognition was not effective before operation or necropsy. Hæmaturia is the most common symptom, occurring in up to 90% of cases. Diagnosis is easily made when the tumour protrudes through the ostium of the sac and is seen cystoscopically; this, however, occurs only rarely. It is important to study the cystograms very carefully and, wherever possible, to inspect the interior of the diverticulum endoscopically. It is probable that radical diverticulectomy has been employed too little in the past, and it is well to remember that in this disease the survival rate varies in inverse proportion to the degree of infiltration of the sac wall by the neoplasm.

Nephrocalcinosis.

J. D. MORTENSEN AND J. L. EMMETT (*J. Urol.*, April, 1954) define as nephrocalcinosis that diffuse renal parenchymal calcification that can be demonstrated radiographically. Under this definition they have collected 48 cases of this disease from the literature and another 43 from the files of the Mayo Clinic. Analysis of data from these sources has led to the following observations. The primary disease responsible for the development of nephrocalcinosis is recognizable clinically in about 84% of cases. The commonest causes of nephrocalcinosis are hyperparathyroidism, hyperchloræmic acidosis and chronic pyelonephritis. The disease has no characteristic symptoms or physical findings, and its clinical recognition depends on its radiographic demonstration. Adequate clinical study includes determination of concentration in the blood of calcium, phosphorus, carbon dioxide combining power, chlorides and urea. Proper interpretation of these results indicates the nature of the primary disorder responsible for the nephrocalcinosis. Pathological findings in the kidneys are deposits of calcium, primarily in medullary regions in relation to collecting tubules, and chronic inflammatory changes in medullary interstitial tissues. Degenerative changes occur, including development of large areas of destruction, replacement with calcareous deposits, cystic dilatation and deformity of tubules. Early recognition and prompt eradication of the primary causative disorder before renal insufficiency develops offer the best chance of delaying or avoiding the onset of total uræmia.

Special Article.

A CHILDREN'S CLINIC IN KOREA.

I ARRIVED in Pusan, South Korea, at the beginning of December, 1953, to take over a children's clinic started by Dr. Roland Good, of Melbourne, under the auspices of the "Save the Children Fund", a British relief organization.

Now about three hundred children attend daily at the original out-patient clinic and about 100 at a tent lent by the United States Army. This latter served the refugees of the disastrous fire in November when 30,000 people were rendered homeless. The United States Army built winter huts to accommodate 12,000 of the most needy persons.

Pusan is the great refugee centre of South Korea and has doubled its population in the last few years; it is estimated that there are 500,000 refugees crowded into this port. Many of them were living in shacks built of scrap wood, cardboard and tin. Now these shacks are being ruthlessly demolished and their inhabitants rendered homeless in this rainy season. I have four Korean doctors working under my supervision at the children's clinic. We have a laboratory on the premises where bacteriological, hæmatological, urinary and fæcal examinations can be made. Also we have a pharmacy attached, which is stocked with most of the drugs usually required. X-ray work is done for us by the Swedish Red Cross Hospital and local Korean hospital. I am hoping to obtain my own machine very shortly and thereby extend the radiological side of the work.

In June we completed our first 10,000 history cards, and I analysed the findings from the notes. Nearly one-third of the patients came with cough as the outstanding symptom; of these about 600 were considered to be suffering from pneumonia or bronchopneumonia and were treated with penicillin injections. Frequently penicillin was given for a feverish cold without any clinical evidence of consolidation. Nearly all of these patients made a good recovery, in spite of unfavourable homes or frequently no place in which to sleep except under bridges, on railway stations or in market places. One morning I saw a heap of rags on the clinic stairs which moved as I approached. On investigation I discovered a dirty louse-infested beggar boy with bilateral bronchopneumonia. He was transferred to the Swedish Red Cross Hospital and in three days made a spectacular recovery.

About 50 children showed appearances on the X-ray film suggestive of tuberculosis, and in older children this was confirmed by sputum tests. It was soon evident that hospital accommodation was necessary for many of these tuberculosis patients. Two wards were made available by the Korean Red Cross, and these were soon filled to their maximum capacity of twenty. As the warmer weather approached it was possible to obtain a squad tent from the United States Army, and a dozen of the convalescent children were transferred to this camp at the seaside.

A special tuberculosis clinic is held every Wednesday afternoon and there are about 260 children on the roll. These include children with tuberculous lymphadenitis, bone lesions, peritonitis and meningitis as well as pulmonary conditions.

The results with streptomycin and PAS have been interesting; five patients with extensive bilateral miliary tuberculosis have made dramatic recoveries. Also those with bronchopneumonic involvement have made good progress.

Our experience with tuberculous meningitis has been disappointing. Generally the patients come in with well-established meningitis, and several have died in the first few days of treatment. Others have made slow and incomplete recoveries with residual palsies and mental retardation. Two relapsed after apparent cure and returned some weeks later with paralysis. It seems imperative to keep all patients with tuberculous meningitis on a maintenance dose of streptomycin for nearly two years. Tuberculous lymphadenitis has been seen in the cervical, mediastinal and hilar glands and has usually been treated with bi-weekly injections of streptomycin. I am not convinced that any great benefit is derived from the injections, but as the home conditions of these patients are so unsatisfactory and their diet is insufficient in quantity and quality, I feel that all possible help should be given them, so include powdered milk, cod liver oil and extra foods.

Several girls between twelve and fifteen years of age have adult chronic pulmonary tuberculosis, and they are making no improvement with streptomycin. Spinal tuberculosis is among the most tragic conditions I have seen, and all the 40 patients suffering from it have long-standing lesions, most of which have produced gross deformity with kyphosis. Three have marked paralysis and half of them have discharging sinuses, in two cases draining through the thigh. Those who have not marked kyphosis I have put in plaster jackets to try to prevent deformity.

There is no hospital accommodation in Korea for tuberculous children, and I am hoping to have a 100-bed children's sanatorium built. However, it is estimated that there are nearly 1,000,000 children with tuberculosis in a population of 25,000,000, so the immensity of the problem can be appreciated.

Among the pulmonary lesions 12 cases of *Paragonimus westermani* were proved bacteriologically. This is caused by eating uncooked lobsters from which the fluke finds its way into the lung. The patient presents with a history of cough and blood-stained sputum, which frequently contains the large typical ovum easily recognizable under the microscope. The X-ray appearances are variable, ranging from a definite cystic area to vague irregular densities, and a radiological diagnosis is seldom possible. I understand from Korean doctors that after a prolonged period of cough and blood-stained sputum the patient generally makes a complete recovery. The liver fluke also causes hepatic disturbance, and clonorchiasis is a serious crippling disease and the liver is extensively damaged.

Bowel disturbance has become increasingly common during the moist summer heat. Non-specific diarrhœa is very frequent with all age groups, and in most cases responds very quickly to sulphaguanidine—in infants with feeding adjustments.

In most cases lasting over a week stool examinations have been carried out and 32 cases of amœbic dysentery have been identified. Worm infestation is almost universal in Korean children, and nearly every stool examined is positive for ascariasis and trichuriasis. A course of hexylresorcinol and magnesium sulphate is given and removes many worms. Six patients have presented with signs of obstruction, but a high enema generally clears the obstructing mass of worms and fæces; occasionally worms are vomited, especially after an anæsthetic.

Hookworms are very rarely found and are frequently the cause of anæmia. Santonin seems to be quite effective in expelling these. Gross anæmia and marked vitamin deficiency were obvious in 250 patients. Milder phases due to insufficient and improper diet are exceedingly common.

In the infant welfare clinic all the babies are given vitamins as a routine. Two tragic cases of blindness from vitamin *A* deficiency did not respond to treatment. Numerous cases of œdema due to malnutrition and resembling beriberi have been very disappointing, as several of the patients just collapsed and died after a period of response to treatment. Post-mortem examinations were unobtainable, but I suspect an œdema of the myocardial muscles.

Skin lesions are very prevalent and nearly every boy seems to have ringworm of the scalp made very obvious by the Korean method of cutting the hair very short. Two cases of generalized eruption resulting from a long-standing infected ringworm were very interesting. Eczema of various types was also very common. *Impetigo contagiosa* was obvious in a large proportion of the children. Superficial abscesses very frequently resulted from these skin lesions either locally or in the adjoining glands. These occurred because of lack of cleanliness and neglect of treatment.

Conjunctivitis was present in over 300 patients, and many of the infections were chronic. Discharging ears were found in 800 children, and most of these gave a long history of otorrhœa. It was difficult to assess the degree of deafness, but the percentage with impairment of hearing must be high. Surprisingly few showed any clinical evidence of mastoiditis. Tonsilitis was present in 340 cases, but in only five of these was a bacteriological diagnosis of diphtheria made. This was due to an extensive campaign of diphtheria toxoid inoculation.

Whooping-cough was distressingly prevalent in about 630 children. Owing to the difficulty of obtaining pertussis vaccine no planned campaign for prophylaxis for infants or contacts could be arranged. Small supplies of "Aureomycin" enabled me to try the effect on a few patients, but I was not convinced that it either cut short the period of paroxysmal coughing or lessened the number who developed bronchopneumonia. I hope to obtain more liberal supplies of vaccine and to inaugurate an intensive campaign of prophylaxis. Mumps was seen in only 17 cases, and there were no complications. Measles were present in 120 cases, and many children were brought whose parents associated their present ill health with a recent attack of measles. Several cases of bronchopneumonia and a few of tuberculosis could be linked up with measles.

Chickenpox was present in 56 children and was always mild. In a country where smallpox is endemic it is impor-

tant to differentiate between these two diseases. Only one case of smallpox was diagnosed, and this is a credit to the wholesale vaccination organized throughout the country.

Eighty children were seen suffering from paralysis resulting from poliomyelitis. No treatment had been given these children, and so our physiotherapy was very acceptable to the parents. Three patients with encephalitis were treated and the infections were assumed to be the Japanese K type reported in Japan and Korea. Two survived without obvious residual defects. One girl brought in unconscious with meningitis following a middle ear infection made a dramatic recovery after two million units of penicillin given over forty-eight hours. Congenital syphilis was confirmed by the cardiolipin test in 22 cases, but had the test been applied as a routine I am certain many more would have been found. Twenty of the patients had interstitial keratitis.

Rheumatic conditions, including two cases of rheumatic fever, were found in 30 cases. It was surprising to realize that only two definite cases of rheumatic endocarditis were discovered, in comparison with 18 cases of congenital heart conditions. As we had no facilities to examine these patients with a fluoroscopic screen and certainly no opportunity to prepare arteriograms, it was difficult to decide on the congenital lesion. At least four suggested patent *ductus arteriosus*, and three were typical of Fallot's tetralogy; unfortunately there is no surgeon interested in cardiac surgery in this part of Korea.

Chronic nephritis was diagnosed in 27 cases, and the treatment was unsatisfactory in 25 of them. Osteomyelitis was seen in only 12 children, and ten of these cases were chronic. In one of the acute cases there was involvement of the lower end of the left humerus, the right tibia and the left femur; the condition has been controlled by penicillin and "Terramycin".

Only seven children with fractures were treated, which is an exceptionally small number. A history of epileptic fits was present in eleven children, and all were of long standing. Eleven patients had obvious inguinal herniæ, and two of these were obstructed. They were operated on at a local hospital and made a good recovery. Umbilical protrusions are very frequent in babies, but the majority seem to recede eventually.

Malaria is rare in this area, so only two cases have been diagnosed. Two cases of relapsing fever with typical spirochæte were diagnosed microscopically.

One boy came in suffering from rabies, but unfortunately he was sent away before I could see him and arrange to obtain some serum. Three children with leprosy were seen, and one attends regularly for treatment. There are supposed to be 50,000 lepers in Korea and leprosarium accommodation only for 10,000, and these are booked ahead for two years. The local leprosarium has 250 affected children and 20 still unaffected in spite of living with leprous parents. I am now starting a clinic to treat these children with D.D.S. (diamino diphenyl sulphone) and hope to obtain encouraging results therefrom, as they have never received any treatment.

J. N. BURGESS, M.B., B.S., D.D.R. (Melbourne), M.F.R., M.C.R.
Korea.

British Medical Association News.

MEETING OF THE FEDERAL COUNCIL.

A MEETING of the Federal Council of the British Medical Association in Australia was held at the Hotel Canberra, Canberra, Australian Capital Territory, on October 28, 1954, DR. A. J. COLLINS, the President, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. A. J. Collins, D.S.O., M.C., Dr. W. F. Simmons, Dr. H. R. R. Grieve, Dr. A. J. Murray, O.B.E.

Queensland: Dr. A. E. Lee, Dr. H. S. Patterson.

South Australia: Dr. L. R. Mallen, Dr. C. O. F. Rieger.

Tasmania: Dr. M. W. Fletcher.

Victoria: Dr. H. C. Colville, Dr. Charles Byrne, Dr. Robert Southby.

Western Australia: Dr. C. W. Anderson, Dr. A. D. Wilson.

Dr. J. B. G. Muir, of Tasmania, was prevented from attending the meeting by the grounding of all aeroplanes at Hobart owing to bad weather conditions.

Pensioner Medical Service.

At the meeting of the Federal Council held at Brisbane at the end of August, 1954, a discussion took place on the Pensioner Medical Service. This meeting was reported in the issue of October 16, 1954. At that meeting the General Secretary read replies which had been received from the Branches to certain questions formulated at the previous meeting of the Federal Council. The Federal Council, on receiving these replies at the August meeting, reaffirmed its previous policy in regard to the treatment of pensioners at concessional rates, and decided to convey this decision to the Minister for Health, and at the same time to request the Minister to arrange an interview with the Prime Minister.

Subsequent to the meeting in August, and arising out of the letter to the Minister for Health, proposals were received from the Government in regard to the future of the Pensioner Medical Service. These proposals were submitted to the Branches for their consideration, and at the special meeting of the Federal Council held in Canberra on October 28, 1954, the views of the Branches in regard to these proposals were considered. The Branches were unanimous in their acceptance of the Government's proposals.

It was accordingly decided to inform the Government that, subject to certain provisos, the proposals of the Government were acceptable to the profession for a period of twelve months.

The acceptance of the proposals will mean:

1. That persons, entitled to pensions as a result of the 1954 amendment to the *Social Service Consolidation Act* liberalizing the means test, will be entitled to the pensioner medical service.

2. That the rates for attendance will be increased by one shilling (that is, consultation at the surgery 10s., visit to the home 12s.) as from November 1, 1954.

3. That a committee will be appointed by the Government to consider the future of the Pensioner Medical Service.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal Alexandra Hospital for Children, Camperdown, New South Wales, on June 17, 1954. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital. Part of this report appeared in the issue of November 13, 1954.

Rheumatic Disease in Childhood.

A demonstration was arranged by the Institute of Child Health illustrating the following features of rheumatic disease in childhood: (*a*) Current ideas about salicylate and hormone therapy—a graph was shown illustrating the treatment of a child suffering from severe rheumatic pancarditis with cortisone. (*b*) Recent ideas on prophylaxis.

Pseudohermaphrodites.

PROFESSOR LORIMER DODS and DR. ROBERT VINES discussed the treatment of seven female pseudohermaphrodites whose conditon was caused by congenital adrenal hyperplasia. The discussion was illustrated with photographs and graphs. It was stated that cortisone therapy had caused loss of signs of virilism in the older children and appeared to have checked the development of such signs in the younger children. Two infants of the series had signs of disturbed electrolyte metabolism and required additional salt. One of them needed large doses of desoxycortone in addition to cortisone.

Patent Ductus Arteriosus.

DR. T. Y. NELSON showed a child, aged six months, who since birth had had difficulty in feeding owing to ready exhaustion, pallor, cyanosis and an increased respiratory rate. On examination and investigation, a diagnosis of patent *ductus arteriosus* and congestive cardiac failure was made. The child was digitalized and given appropriate antibiotics, and then operation was carried out by ligation of a patent *ductus arteriosus*. It was noted that there was more periductal inflammation than usual. The post-operative condition was good. Administration of digitalis and antibiotics was suspended on the fifth day after operation. The child was then bright, no murmur could be heard, and the

respiration was only moderately rapid. Post-operative X-ray examination of the chest showed a small pneumothorax and later a collection of fluid, but the cardiac shadow had decreased. Dr. Nelson commented that the early development of congestive heart failure was unusual in cases of patent *ductus arteriosus* and made operation at such an early age imperative if the child was to survive.

Congenital Diaphragmatic Hernia.

DR. J. STEIGRAD presented a girl, who had been born at 5.30 a.m. on April 13, 1953. Cyanosis was present at birth and continued until relieved by oxygen; even then the respirations remained rapid and laboured. X-ray examination showed a large left diaphragmatic hernia with air-containing bowel almost entirely filling the left thoracic cavity. The child was transferred to the Royal Alexandra Hospital for Children on the same day, and X-ray examination confirmed a defect in the left hemidiaphragm with herniation of the mid-gut loop into the left hemithorax. There was associated displacement compression and atelectasis of the lungs, and a mediastinal shift. At 7.12 p.m. the child suddenly collapsed and went black. Relief was obtained by forcing oxygen into the mouth under pressure and by intubation and forced insufflation. At 8 p.m. laparotomy was carried out, the viscera were returned to the abdomen, and the pleuroperitoneal canal was repaired with two layers of silk sutures. Withdrawal of the bowel produced immediate respiratory relief. After a rocky convalescence, the child's condition rapidly improved, and she was discharged from hospital on April 28, 1953. At a follow-up examination in September, 1953, the child was well. Five days later, however, she was readmitted to hospital with the story that she became breathless when she moved around or became excited. She was otherwise well. On examination of the child, the trachea was found to be deviated to the right side, and there was dullness at the left lung base. The child looked very well. X-ray examination revealed a recurrence of the left diaphragmatic hernia. At operation on the following day a sac of peritoneum containing large bowel was reduced, and the diaphragm edge was sutured with 28 gauge wire around the ninth, tenth and eleventh ribs. Post-operative bronchopneumonia responded to penicillin, and convalescence was otherwise uneventful. Subsequently the child progressed well without recurrence of the trouble.

Another child presented by Dr. Steigrad had been born on September 4, 1953. The delivery was normal, but the child did not breathe or cry readily at birth. The heart sound was heard to the right of the mid-line. X-ray examination revealed bowel in the left hemithorax. On admission to the Royal Alexandra Hospital for Children fourteen hours after birth, the child was mildly cyanotic, even when receiving oxygen, and respiration was faintly grunting. Examination revealed that the mediastinal structures were deviated to the right side. At operation, which was performed almost immediately, small gut, large gut and spleen were returned from the thoracic cavity to the peritoneal cavity, and the defect in the diaphragm was repaired in two layers. On the following day, grunting respiration returned, and X-ray examination showed a tension pneumothorax with a considerable mediastinal shift to the right side. This responded satisfactorily to the insertion of a tube via a trocar and cannula, the tube being connected to underwater drainage. The patient was discharged from hospital fit eleven days after operation, and there was no subsequent recurrence of trouble. The patient was now well and healthy.

Fractured Skull.

Dr. Steigrad also showed a girl, aged three and a half years, who had been admitted to hospital in January, 1954, with a grossly comminuted fracture of the right parietal bone, resulting from an accident eleven days previously. The child was conscious and cooperative, and no abnormalities of the central nervous system were evident. X-ray examination confirmed the diagnosis, the region as a whole being depressed. One fragment was impacted into the subjacent brain. At craniotomy, the depressed fractured area of the skull was raised, and convalescence was uneventful.

Skull Hæmatoma.

Dr. Steigrad's next patient was a boy, aged two years and four months, who had been admitted to hospital in April, 1954, with a fractured skull, the result of a fall of seven and a half feet on to hard ground. No loss of consciousness had occurred immediately, but the child became unconscious one hour later. When examined on admission to hospital, the child was unconscious and had a large hæmatoma of the left parietal region. The abdominal reflexes and all deep reflexes were absent. Babinski's sign was present on both sides, and occasional spontaneous movements of the limbs were noted. X-ray examination showed a linear fracture of the left parietal bone extending almost from the base to the vertex of the skull. The fragments were not depressed. The same night, generalized spasticity of the limbs occurred, and the left pupil was found to be larger than the right. Cheyne-Stokes respiration began at 11.45 p.m. Operation was performed, and through a burr hole a large extradural hæmatoma was evacuated. The child regained consciousness and recovered completely.

Crohn's Disease.

Dr. Steigrad then showed a boy, aged eleven years, who had been operated on for acute appendicitis, but at operation free blood-stained fluid was found in the abdominal cavity and a large appendiceal mass, retroperitoneal and retrocaecal in position, was delivered into the wound and removed. The appendix appeared hæmorrhagic and gangrenous. The pathologist reported suppurative inflammation of the mucosa, the muscular wall being largely replaced by fibroblastic granulation tissue with numerous follicles composed of lymphoid and reticular cells and an occasional giant cell of foreign body type. The diagnosis was Crohn's disease. The child was discharged from hospital six weeks after operation, feeling well.

Diaphragmatic Hernia with Obstruction.

Dr. Steigrad's next patient had been admitted to hospital in January, 1954, at the age of seven and a half months with a history of listlessness, anorexia and vomiting for two days. A deep resistance was found in the hypochondrium, vomiting became more copious, and bowel wash-out revealed a small amount of firm stool with a flake of blood and mucus. X-ray examination showed the presence of loops of bowel in the left hemithorax, and at operation most of the stomach and omentum, part of the transverse colon and considerable lengths of ileum were found to have passed through a small defect in the left anterior part of the diaphragm. These were delivered, and, although the omentum and several loops of the small gut showed signs of early strangulation, they were considered to be viable. Convalescence was satisfactory.

Gunshot Wound to the Neck with Spinal Shock.

Dr. Steigrad then showed a boy, aged six and a half years, who had been admitted to hospital in October, 1953, two hours after receiving a gunshot wound of the neck. The bullet had entered the left side of the jaw near the angle of the mandible. When found by his father, the boy could not speak or move. On the way to hospital he was able to talk in a whisper and had small movements of the right leg and right arm. On admission to hospital, the child was found to have no movement in the left arm, left side of the chest, left side of the abdomen and left leg. Very slight movement of the right arm was present. The cranial nerves were intact, except that no shrugging of the shoulders was noted. All limbs were flaccid, all deep reflexes on the left side of the body were absent. Examination for Babinski's sign showed that movement of the great toe was upwards on the right side and downwards on the left. On the same afternoon, left lower facial paresis was noted. No diaphragmatic movements were then evident, anæsthesia was present in the right arm and hand, and the great toe was upgoing on both sides. X-ray examination showed the presence of a metallic foreign body between the spinous processes of the first and second cervical vertebræ. Some debris was seen anterior to the foreign body, but no bony injury was detected. The child developed swelling in the region of the wound. Movement of the limbs and left side of the abdomen and chest gradually returned, and the left facial paresis gradually disappeared. The sensation in the right arm and hand improved slowly. Treatment was given with physiotherapy and a temporary Jones's frame, and in December the bullet was removed from the first intervertebral space three-quarters of an inch to the right of the mid-line. When discharged from hospital in March, 1954, the child was walking well.

Congenital Fibrosarcoma of the Left Forearm.

DR. E. H. GOULSTON showed a girl who, when born on October 26, 1951, had had a firm swelling of the distal part of the left forearm. On November 23, 1951, she was transferred to the Royal Alexandra Hospital for Children, where X-ray examination showed considerable swelling of the soft tissues of the lower end of the forearm with narrowing and deformity of the lower end of the ulna suggesting a pressure defect rather than bony involvement. Biopsy examination of the fleshy tumour of the left forearm was carried out, and the pathologist reported the diagnosis of fibrosarcoma. He stated that local malignant change was undoubted, and the possibility of wider dissemination could not be excluded. X-ray examination of the chest showed no abnormalities in

the lung fields and thoracic cage. On December 4, amputation was performed at the level of the left mid-humerus. From examination of the tissue removed at operation the pathologist reported that the exact origin of the growth could not be determined with certainty, but it appeared to arise from fibrous tissue and merged with the wrist joint capsule, though the joint space was not penetrated. There was likewise no penetration into the bone, though the cortex lying beneath the growth showed active lacunar resorption. The histological characters of the growth were similar in all parts and did not depart in any way from the portion of tissue previously removed and examined. There was no evidence of dissemination beyond the main mass in the part amputated. A course of deep X-ray treatment was commenced at the Royal Prince Alfred Hospital on December 21, 1951. Subsequently the child had remained well, and there had been no evidence of any metastatic spread.

Congenital Tracheo-Œsophageal Fistula.

Dr. Goulston showed three patients who had been suffering from congenital tracheo-œsophageal fistula, without atresia in two cases and with atresia in one. In each case the fistula had been successfully dealt with at operation and the child had subsequently done well. Œsophageal dilatation had been necessary in the case associated with atresia.

Cat Scratch Disease.

Dr. Goulston with Dr. Steigrad also presented details of three cases of cat scratch fever. In the first case, the child had been admitted to a country hospital in August, 1953, with a diagnosis of pyrexia of unknown origin. He then had cervical axillary and inguinal lymphadenopathy and complained of pain in the right hip. Extensive subsequent investigation produced negative findings until examination was made of a lymph gland excised from the left axilla. The pathologist reported that the specimen consisted of remnants of altered lymphoid tissue with granulomatous extensions into the surrounding adipose tissue. The outstanding feature was the presence of a number of necrotic foci of varying size and shape. Those were surrounded by a narrow wall of deeply eosinophilic epithelioid cells, some of which were multinucleated. The majority of the necrotic foci were converted into abscesses by accumulations of polymorphonuclear cells, and similar cells were abundant in the intervening non-necrotic parts. The diagnosis was a non-bacterial suppurative and necrotic lymphadenitis consistent with cat scratch infection.

The second child had a history of having been born with a lump in the right mandibular region which had grown to the size of a small egg. This had been excised. Another lump of the size of a walnut was now present in the same area. It was firm, not hot or tender, of smooth contour and freely mobile, and appeared to be subcutaneous. The lump was removed and found to be a firm, pale lymph gland of normal architecture and containing no cysts. There were, however, certain very distinctive histological changes in the gland, both within the follicles and in the lymph cords, and the pathologist stated that though those varied from part to part, they seemed to be only varying stages of the same process. In what were probably the more recent foci, there was a central focus of fibrin and polymorphonuclear cells, and that was surrounded by a wide zone of epithelioid cells mixed with polymorphonuclear cells and plasma cells. In older foci, the central leucocytic focus was absent and the follicular accumulation of epithelioid cells was smaller, often with giant cell forms, while in still other areas there were the beginnings of connective tissue proliferation. The comment was made that it seemed unlikely that the histological lesions in the gland were the result of a tuberculous infection. Unfortunately, the patient was discharged from hospital before the result of the Mantoux test was read. However, the changes were consistent with the diagnosis of cat scratch adenitis.

The third child had been admitted to hospital in February, 1952, for lymph gland biopsy of the left groin, the mother having noticed lumps in that area fifteen months previously when washing the child during convalescence from measles. The results of the biopsy were inconclusive. Chest examinations and Mantoux tests revealed no abnormality. In December, 1952, the child apparently developed glandular fever. In January, 1953, a further biopsy of a gland from the right axilla was carried out, and in March, 1953, another gland was removed from the left groin. Direct examination of material from the glands and attempted culture produced no evidence of *Mycobacterium tuberculosis*, and the child's chest remained radiologically normal. The pathologist reported that the glands removed in January, 1953, contained epithelial follicles. These were large and numerous in the largest gland, where caseation was a prominent feature in the centres of the follicles. Langhan's giant cells were not infrequent in these larger caseous foci. The diagnosis was made of tuberculous lymphadenitis. In the gland removed in March, 1953, the follicles were found to be enlarged and the sinuses dilated and packed with histiocytes and a smaller number of polymorphonuclear cells. No tuberculous lesions were present. The diagnosis was made of acute non-specific lymphadenitis consistent with cat scratch fever.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE LUNATIC ASYLUM.[1]

[From the *Australian Medical Journal*, June 1, 1847.]

DURING last session of the Legislative Council a select Committee was appointed "to enquire into the management of the Lunatic Asylum, Tarban Creek, with instructions to take evidence and report whether the system therein adopted requires modification and how far the buildings are suitable or sufficient for the proper treatment and classification of the inmates". This report was given in our number for December last and on the 11th inst. pursuant to notice Mr. Cowper moved that the report be transmitted to the Governor with an address from the Council requesting that "he will carry into effect the recommendations of the Committee as speedily as circumstances will admit". These recommendations involve an immediate outlay of £8000 or £10,000 for alterations or additions to the buildings and a further annual expenditure of £1000 or £1200 for improvements in the management. The present building was originally intended to contain only 60 inmates, but as many as 175 had at one time been placed there and the number now is 150, while there are 100 in the Hospital at Liverpool and some also in the Factory at Parramatta. "Now", as was very properly observed by Mr. C., "it is utterly impossible that a building originally for the reception of only 60 persons can with any degree of comfort or classification contain 150." It is preposterous—the idea would be repudiated even as regards criminals of thrusting 150 into a space only calculated for 60. We shudder to contemplate the state of these poor creatures under such circumstances. Coercion must necessarily take the place of supervision, and if any are ignorant of the effects of such a system let them read attentively the lectures of Dr. Conolly on the subject now in course of publication in this Journal. Mr. Windeyer opposed the motion on the ground of the expenditure involved in it for new buildings while there were so many already in existence which might ultimately be converted to this use as Hyde Park Barracks, the Factory and Gaol at Parramatta and the gaols in other parts of the colony, at the same time that he talked in terms of praise of the recommendation in the report with regard to the accommodation and classification of the patients and agreed that the "Evidence was quite sufficient to prove that the present system wanted reformation". This blowing hot and cold water with the same breath—this confusion of ideas between humanity and parsimony—this attempt to combine an improved system of management and classification with the refusal of the means to carry it out—shows how little the honourable member knows of the subject of which he talks, otherwise he would be aware that such places as he speaks of, built for the safe keeping of criminals with their high walls, their confined and gloomy courtyards, their "chevaux de frize" and stone cells are totally incompatible with such a system. In the same parsimonious spirit Mr. W. opposed the proposition for paying the inspectors to be appointed and talked in terms of the most virtuous indignation against what he called the "sweeping doctrine that has been laid down that nothing could be done here that was not paid for" and put forward the Council as an example to the contrary. Why the "doctrine" is only a leaf out of Mr. W.'s own book for he himself has been known heretofore to protest against labour without pay. But that was in matters connected with his own profession and he does not choose to remember that the only circumstances which reconcile himself and others of the Council to their unpaid services are the honour and patronage attached to them.

[1] From the original in the Mitchell Library, Sydney; abridged.

Special Correspondence.

NEW ZEALAND LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Obituary: Thomas William James Johnson.

T. W. J. JOHNSON, who died in Auckland on October 16, 1954, at the age of sixty-nine years, had been senior physician to the Auckland Hospital, examiner in medicine to the University of Otago, and a member of the Medical Council. He was a Fellow of the Royal College of Physicians of London, as well as a Foundation Fellow and Vice-President of The Royal Australasian College of Physicians, and took a leading part in many organizations and activities connected with Auckland Hospital.

He entered the University of Otago as a Junior Entrance Scholar from Thames High School and Auckland Grammar School. During his medical course he won every prize offered, including the Medical Travelling Scholarship, as well as getting his hockey blue representing Otago at hockey and being president of the Students' Association.

He held resident posts at Dunedin Hospital and at the Middlesex and Maida Vale Hospitals. He carried on general practice in Napier for ten years, and after a further period abroad settled in Auckland as a consulting physician; there he soon acquired a large practice and was widely known for his diagnostic acumen.

In private life he was a keen angler and golfer and student of Maori history, lore and language. He was an authority on native New Zealand flora and fauna. Once asked by a distinguished English physician visiting this country why he did not stay in London as he had been asked to do many times, he replied: "I thought of your grey skies and our blue ones, so I chose the blue."

Dr. Johnson is survived by his widow and twin sons, Maurice and Alan, who both practise in Auckland.

Correspondence.

EAR COMPLICATIONS ASSOCIATED WITH SPEAR FISHING.

SIR: In his article "Ear Complications Associated with Spear Fishing", Dr. D. G. Perrett advises that skin divers should use ear plugs. This is the antithesis of the advice of Captain Cousteau, the inventor of the "Aqualung" and a world authority on the subject. On page 3 of his book, "The Silent World", speaking of his early experiences, he says: "I swallowed, as one does when landing in an aeroplane, to open my Eustachian tubes and stopped the pain. (I did not wear ear plugs, a dangerous practice when under water. Ear plugs would have trapped a pocket of air between them and the eardrums. Pressure building up in the Eustachian tubes would have forced my eardrums outward, eventually to the bursting point.)"

The pastime of skin diving is popular, so that it is important for the profession to give the correct advice; the problem is essentially one of establishing pressure equilibrium between the water and the organism as a whole.

For anyone wishing to read an enthralling true thriller, they should beg, borrow or steal (but not my copy) Cousteau's fascinating book, which contains some of the most exciting experiences and extraordinary photographs ever published.

Yours, etc.,

F. S. HANSMAN.

143 Macquarie Street,
Sydney,
November 9, 1954.

SIR: I wish to thank Dr. D. G. Perrett for his report in THE MEDICAL JOURNAL OF AUSTRALIA, October 30, 1954, page 709, of the ear damage sustained by two spear fishermen. However, I would like to point out that his advice to followers of this and allied underwater activities is misleading in two respects. Ear pads or plugs of any sort should not be worn in underwater diving. The reason is simple: in order to avoid damage to the tympanic membrane and middle ear, the pressure on each side of the eardrum must be able to equalize when diving at varying depths. This can only be achieved by avoiding obstruction in both the Eustachian tube and the external auditory canal. Ear pads could act as an obstruction to the latter. This is the teaching of the medical advisers to the Underwater Research Group of Queensland, and is readily demonstrated. Descent to a few feet under water with one ear hole occluded results in discomfort increasing to pain in the ear unless, perchance, the Eustachian tube is also blocked, as, for example, during the course of a "cold" or other naso-pharyngeal sepsis. A similar descent with an unobstructed external auditory canal is entirely free of discomfort provided again the Eustachian tube is patent.

The wearing of breathing apparatus such as an "Aqualung" does not alter the pressures on the eardrum and is therefore no protection against ear damage when diving.

Yours, etc.,

S. R. STEVENS.

553 Brunswick Street,
New Farm,
Brisbane.
November 12, 1954.

SUBCUTANEOUS INJECTION OF ADRENALINE DURING ADMINISTRATION OF A GENERAL ANÆSTHETIC.

SIR: The infiltration of an operation site with a dilute solution of adrenaline during the administration of a general anæsthetic is a valuable way of decreasing bleeding. There are a number of points in this procedure which are important.

The decreased bleeding is brought about by the vasoconstrictive action of adrenaline and the distension of the tissue with fluid. Adrenaline so injected is destroyed in part locally and is partly absorbed into the circulation, where it may produce measurable effects upon heart rate and rhythm and upon the blood pressure. With more concentrated adrenaline solutions the proportion absorbed into the circulation is greater than with dilute solutions. Observation suggests that with a dilution of 1:200,000 there is good vasoconstriction with minimal effects upon the circulation, but with dilutions of 1:80,000 and stronger the systemic effects are much greater with only very little improvement in ischæmia.

The circulatory effects of procaine and "Xylocaine" tend to be opposite from those of adrenaline, so that the effect of combining the two should be a reduced risk of toxic reactions from either.

The following suggestions are made: (i) Use adrenaline out of ampoules only; this ensures potency and sterility. (ii) Measure with a one cubic centimetre syringe; measuring with an eye dropper or guessing amounts is dangerous when one is dealing with potent drugs. (iii) Regard a dilution of 1:200,000 as the one for general use, and 1:100,000 as the maximum strength. (iv) Dilute the adrenaline with 0·5% procaine or 0·5% "Xylocaine" in saline rather than with saline.

These views on adrenaline are not new. They were originally stated by Braun, and elaborated by Pitkin.

Yours, etc.,

W. H. J. COLE.

342 Albert Street,
East Melbourne,
October 20, 1954.

Medical Practice.

MEDICAL PRACTITIONERS ACT, 1938-1953.

THE following proclamation has been published in the New South Wales Government Gazette, Number 181, of November 5, 1954:

I, Sir John Northcott, Knight Commander of the Most Distinguished Order of Saint Michael and Saint George, Knight Commander of the Royal Victorian Order, Companion of the Most Honourable Order of the Bath, Lieutenant-General on the Retired List of the Australian Military Forces, Governor of the State of New South Wales and its Dependencies in the Commonwealth of Australia, with the advice of the Executive Council and in pursuance of the provisions of the Medical Practitioners Act, 1938-1953, do, by this my Proclamation, declare that the Proclamation published in the Government Gazette on 4th December, 1953, suspending the operation of subsection (3) of section

17 of such Act for a period of twelve months from the 1st January, 1954, is hereby revoked and cancelled on and from the 10th November, 1954.

Signed and sealed at Sydney, this twenty-seventh day of October, 1954.

By His Excellency's Command,

M. O'SULLIVAN.

The subsection referred to is as follows:

(3) Where any person is registered under this Act pursuant to this section the following provisions shall have effect:

(a) The registered person shall be:

(i) competent to accept or to hold any appointment as a medical officer in any public hospital or separate institution within the meaning of the Public Hospitals Act, 1929-1937, or as a medical officer in any private hospital or other institution approved by the Board;

(ii) qualified to sign any medical certificate of the cause of death of any deceased person where such certificate is given in the course of his duties as such a medical officer.

(b) Except as provided in paragraph (a) of this subsection no such person shall practise his profession unless and until:

(i) he has, for a period of twelve months, or for periods amounting in the aggregate to twelve months, served as a medical officer in one or more of the hospitals or institutions referred to in subparagraph (i) of paragraph (a) of this subsection; or

(ii) he satisfies the Board that he has, for a period of twelve months, or for periods amounting in the aggregate to twelve months, served as a medical officer in one or more hospitals or institutions (whether in New South Wales or elsewhere) approved by the Board.

(c) If the Board is satisfied that the registered person is unable by reason of physical incapacity to accept or to hold any such appointment as is referred to in subparagraph (i) of paragraph (a) of this subsection it may agree to accept, as the equivalent of the period of service referred to in paragraph (b) of this subsection, proof that the registered person has, for a period of twelve months or for periods amounting in the aggregate to twelve months, received special tuition of a nature and type approved by the Board in some branch of medicine or surgery approved by the Board.

Where the Board is satisfied that the registered person has complied with the requirements of this paragraph the registered person shall not be precluded by paragraph (b) of this subsection from practising his profession.

(d) Any person who practises his profession in contravention of the provisions of this subsection shall be deemed not to be registered.

This subsection shall not apply to or in respect of any person registered upon proof of the matters referred to in paragraph (d) or paragraph (e) of subsection one of this section or in subsection two of this section.

This subsection shall not apply to or in respect of any person registered before the first day of January in the year one thousand nine hundred and forty-one.

Obituary.

FREDERIC WOOD JONES.

We are indebted to Professor S. Sunderland and Associate Professor K. F. Russell for the following appreciation of the late Professor Frederic Wood Jones.

Frederic Wood Jones died in London on September 29, 1954.

He was one of the leaders in contemporary scientific thought, not only in his own field of human anatomy and physical anthropology, but also in the wider field of comparative vertebrate morphology, his inquiries ranging over the whole of that field of life in which he regarded man as only the central figure. In fact he maintained a broad biological outlook from the beginning of his working life until the end. This, together with his insistence on the comparative and functional approach to the study of structure, has led to his being recognized as the nearest approach to John Hunter, for whom he entertained the greatest admiration. His association, in the formative years of his career, with such figures as Arthur Keith and Elliot Smith almost certainly influenced his early thinking and directed his initial endeavours into the evolution of man. Much of his work in this field was concerned with the probable path by which the primates and man originated. His main thesis that man may have separated from the rest of the primate stock in the pre-simian stage of evolution, is one that can be reasonably argued in view of the highly speculative nature of much of the existing information on this subject. His writings also directed attention to the need for further information and the urgency for revising the vague and undefined nomenclature commonly employed by evolutionists, defects that were, to a degree, responsible for much of the controversy regarding the origin of man.

Throughout his life he remained a staunch Lamarckist and with great dialectical skill sustained this thesis in a number of his publications. His vigorous defence of the teleological approach in biology and the inheritance of acquired characters exasperated many, but his persistence and the depth and range of his knowledge of the subject commanded respect, while his views were expounded with a lucidity and charm which were both envied and admired. His iconoclastic approach to a controversial subject that can still be argued legitimately was refreshing. Above all, he did not regard evolution as being a simple process, but believed that many factors combined in effecting change. He preferred to regard design and purpose as determining factors rather than chance and chaos, and the case which he makes in support of his belief cannot be dismissed lightly.

He had a profound influence on the teaching and research into human and comparative anatomy, an influence which commenced in the early years of this century and, it is true to state, has not ceased with his death. Such was his force of character and enthusiasm towards the work of his juniors that those anatomists who were fortunate to have him as a mentor owe him a debt that they can never repay and will carry on his teaching and tradition.

To be with Wood Jones in conversation or discussion, to hear him draw on his inexhaustible fund of knowledge, to sample his impish sense of humour and his profound, if at

times trenchant, wit, to hear his keen analysis of some particular piece of research were experiences that will never be forgotten. His friendly, lovable nature and his interest and delight in the work of his juniors have left an indelible mark in every department with which he was associated. He was not slow to criticize statements or papers that he knew to be wrong, but his criticism was friendly and at times jocular.

Many of the views that he held were unorthodox, a fact he himself was the first to admit. In the introduction to "Trends of Life", which was to be his last book, he writes:

> It is inevitable that even the most ordinary of men must, at the end of a long life spent in observing living things, have formed some ideas as to life and its potentialities, its ordering and its probable changes under varying conditions. Such observations and the deductions made from them will of necessity be weighed against the dicta that are considered at the moment to be the pronouncements of orthodox science. But I am not concerned with the current dicta of science that, in a disturbed and transitory period, are held to be orthodox.

The fact that his views were unorthodox was at once stimulating and provocative, and he took great delight in the subsequent discussion and criticism.

Wood Jones was born in London on January 23, 1879, and studied at the University of London, where he took a B.Sc. in 1903. In 1904 he graduated M.B., B.S., and took the conjoint diploma, and in June of the following year he left England to take up the position of medical officer to the cable station on the Cocos-Keeling Islands, where he worked until September, 1906. While there he carried out an intensive research into the formation and growth of coral atolls, which was later published and remains, even today, a standard work on the subject. It was during this period, too, that he met the charming and gracious lady who was to be his wife, Gertrude Clunies, the daughter of George Clunies-Ross. After a brief return to England he was appointed anthropologist to the Egyptian Government in 1907 to assist Elliot Smith in the archæological survey of Nubia, a task which was completed in 1909 and the results of which were published in a series of volumes between 1907 and 1910. Elliot Smith held him in high regard, and in a letter sent to Arthur Keith he wrote: "What a mess there would have been in Nubia if I had not Wood Jones! For I know no other man who could have tackled the stupendous task there as he has done."

From 1909 to 1910 he was lecturer in anatomy at the University of Manchester. From there he was appointed demonstrator in anatomy at both the London and St. Thomas's Hospitals (1910-1912), and in the latter year was appointed to his first chair as Professor of Anatomy at the London School of Medicine for Women, from which he resigned in 1919. During the war period he was a captain in the Royal Army Medical Corps and did a considerable amount of clinical work on nerve injuries and trick movements at the Special Military Surgical Hospital, Shepherd's Bush. His work was published in the *Journal of Anatomy*, and the lectures that he gave at Shepherd's Bush formed the basis of "The Principles of Anatomy as Seen in the Hand", which first appeared in 1920 and is one of his most important books. During the war years, too, he was Arris and Gale Lecturer at the Royal College of Surgeons (1914, 1915, 1916 and 1919). The lectures given in 1915 and 1916 were expanded and published as "Arboreal Man".

In 1919 he made his first contact with Australia when he was appointed to the Elder Chair of Anatomy at the University of Adelaide in succession to Archibald Watson. Wood Jones became very fond of Australia and Australians, a fondness which was to bring him back some years later and which he never lost.

Late in 1926 he resigned his chair at Adelaide and accepted the appointment as Rockefeller Professor of Physical Anthropology at the University of Hawaii, where he worked from 1927 to 1930. In this latter year he returned to Australia, this time to Melbourne, where he was appointed to the chair of anatomy, which had become vacant on the retirement of R. J. A. Berry. During 1932 and 1933 he obtained leave from Melbourne to go to China as temporary director of the Peiping Union Medical College.

He resigned from the Melbourne chair in 1937 and was appointed Professor of Anatomy at Manchester, where nearly thirty years before he had held his first academic appointment. He remained there until 1945, when he became the first Sir William Collins Professor of Human and Comparative Anatomy and Conservator of the Anatomical Museum at the Royal College of Surgeons. In 1947 he was again Arris and Gale Lecturer. When, in 1951, the Royal College of Surgeons entered into an agreement on teaching with the University of London, he was forced to retire from this position because of his age and was instead appointed Honorary Conservator of the Hunterian Collection, a position he occupied until his death.

It is only natural that during his life Wood Jones received many honours. He was awarded the D.Sc. of the University of London in 1910 for his work on the coral atolls and received the same degree from the Universities of Adelaide and Melbourne.

He was appointed a Fellow of the Royal Society in 1925 and a Fellow of the Royal College of Surgeons in 1930. In 1935 he received the Fellowship of the Royal Australasian College of Surgeons. He was also a Fellow of the Zoological Society.

For some years before his death he was a Hunterian Trustee of the Royal College of Surgeons. In 1949 the College awarded him their Honorary Medal, the highest honour they could bestow.

He was appointed the Thomas Vicary Lecturer for 1954, but unfortunately his illness prevented him from delivering the address. The award of this lectureship had pleased Wood Jones, for all his life he had been interested in the history of anatomy and possessed what was probably one of the finest privately owned collections of early anatomical texts in existence; this was sold several years before his death. He had a deep regard for John Hunter and his works, and he took a great joy in the rebuilding of the Hunterian Collection at Lincoln's Inn Fields when he went there in 1945. The Hunterian Collection had been partly destroyed in 1941 and the rest dispersed, and it was Wood Jones who supervised the return of the scattered portions of the great collection, reclassified the specimens and replaced as many of the lost portions as were possible. Many of the replacements came from his own collection of human and comparative osteology, and one has only to peruse the Scientific Reports of the College in the post-war years to appreciate the extent of his generosity. His years as Conservator and Trustee of the Hunterian Museum were probably the period in his life that pleased him most, and when all is said and done, only Wood Jones could have done it in the true Hunterian tradition.

His writings were as diverse as they were extensive. Some appeared in book form, and a list of these is appended to this appreciation; very many more were published in the scientific journals. Many of his papers were written for the non-scientific public, such as his delightful "Unscientific Essays" and "Unscientific Excursions"; some were for children like "Sea Birds Simplified"; others, such as his books on the hand and the foot, immediately became textbooks for the student of anatomy whether undergraduate or post-graduate. During his last year some of his characteristic articles of popular appeal appeared under the pseudonym of "Natterjack". He had a deep knowledge of the sea birds, which was gained during his ocean travels, particularly at Cocos-Keeling, but most of it was gained in Melbourne, when in the long vacation he would tour the Victorian coast on the lighthouse supply ships calling in on all the small islands. During other vacations he would take a group of students and camp on some uninhabited island off the coast and conduct a biological survey on behalf of the McCoy Society, of which he was founder and generous benefactor. There was never a shortage of volunteers when one of these camping holidays was planned.

He was very interested in and had a deep knowledge of the Australian aborigines and did much to assist them in the improvement of their position.

Wood Jones possessed outstanding gifts as a teacher and orator. He lectured with a rare lucidity and fluency and with an enthusiasm that was infectious. This eloquence, together with his profound knowledge of anatomy and the novel and original way in which he dealt with it, and last, but not least, the instructive, skilfully created diagrams with which his lectures were illustrated, made him a brilliant and inspiring teacher.

This spirit has been preserved in "Matrix of the Mind" and in his books on the hand and the foot, which have provided generations of students with many suggestive hints and unusual points of view and represent a refreshing departure from the stereotyped approach of other texts.

As a public speaker he was much in demand, for he had that wonderful capacity of talking about science in a way that the public could understand and enjoy. On every occasion on which he delivered a public address in Melbourne our largest lecture theatres were crowded to the doors, and for his valedictory address the Melbourne Town Hall was packed. There have been few contemporary orators in any walk of life in this country who could exert the

same magnetic influence on both lay and professional audiences with such consistency. His many friends subscribed to a portrait when he was leaving Melbourne; this was painted by McInnes and is now in the Royal Australasian College of Surgeons. The best photograph, however, is the one taken by Julian Smith and is here reproduced.

This account of the career of a remarkable man will do little to enable those who did not come under the spell of his unique personality to picture his outstanding characteristics. *He was possessed of a restless curiosity, was never* repressed in expression, nor did he ever sit on the fence; he was always writing and saying something unusual, and was *always a little apart from the herd. But those whose* good fortune it was to know him need no reminders. By the intense thoroughness of his teaching, by the philosophical *breadth of his outlook and by his own personal* character he wielded an immeasurable influence on many hundreds of medical graduates and students in this country and abroad; and everywhere he was regarded with admiration, esteem and affection.

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Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 63, of October 28, 1954.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Surgeon Commander Eustace Alwynne Rowlands, O.B.E., is promoted to the rank of Surgeon Captain, dated 27th September, 1954.—(Ex. Min. No. 106—Approved 19th October, 1954.)

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 1/55108 Lieutenant-Colonel D. C. C. Sword is confirmed. 1/55108 Lieutenant-Colonel D. C. C. Sword is placed upon the Retired List (1st Military District) with permission to retain his rank and wear the prescribed uniform, 30th June, 1954.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/130106 Captain (provisionally) H. L. Thompson is seconded for post-graduate studies in the United Kingdom, 1st June, 1953.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 30, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5	1(1)	6(5)	..	1(1)	..	..	..	13
Amœbiasis	..	..	..	1(1)	..	..	..	..	1
Ancylostomiasis	..	..	1	..	..	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1(1)	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(3)	4(3)	14(14)	..	..	..	..	..	22
Diphtheria	10(8)	..	7(1)	..	1(1)	..	..	..	18
Dysentery (Bacillary)	..	..	3(3)	2(2)	..	..	..	..	5
Encephalitis	..	..	..	3(3)	..	..	..	1	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	1	..	..	..	..	..	..	1
Hydatid	..	..	..	..	2	..	..	3	140
Infective Hepatitis	79(44)	56(45)	..	..	..	..	..	..	..
Lead Poisoning	..	..	..	..	..	..	1	..	1
Leprosy	..	..	..	..	..	..	..	..	2
Leptospirosis	..	..	2(1)	..	..	..	..	..	1
Malaria	..	..	1(1)	..	..	..	..	..	7
Meningococcal Infection	4(3)	..	2(1)	..	..	1(1)	..	..	1
Ophthalmia	..	..	..	..	1(1)	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	23
Poliomyelitis	1	7(3)	7(1)	7(4)	1	..	..	..	2
Puerperal Fever	1	..	1	..	..	..	..	..	51
Rubella	..	25(7)	..	..	26(22)	..	..	..	..
Salmonella Infection	..	..	..	..	..	..	..	..	66
Scarlet Fever	11(5)	38(27)	8(4)	7(5)	1	1(1)	..	..	..
Smallpox	..	..	..	..	..	..	..	..	1
Tetanus	..	..	1	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	113
Tuberculosis	54(34)	35(24)	4	6(5)	7(6)	4(2)	3	..	..
Typhoid Fever	..	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	1	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

2/111127 Captain R. H. Higham is appointed from the Reserve of Officers, 6th August, 1954. 2/165473 Captain (provisionally) J. E. D. Goldie relinquishes the temporary rank of Major, 30th June, 1954.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 3/129041 Captain B. C. A. Stratford is confirmed. 3/111489 Captain P. J. Robinson is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 5th August, 1954.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

2nd Military District: To be Honorary Captains.—Colin Harrison Mansfield, 26th July, 1954, Philip Francis Cappe, 30th August, 1954, and Edwin William Carr, 13th September, 1954.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course Suitable for Candidates for Primary F.R.A.C.S. Examination.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that it is proposed to conduct a course of lectures suitable for preparation for the Primary F.R.A.C.S. examination to be held in September, 1955. The course will extend over a period of twenty-four weeks from March 7 to August 19 and will consist of two lectures per week, held in the late afternoons. Details concerning fees will be announced shortly. Those interested in attending are asked to register with the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic address: Postgrad, Sydney.

SEMINARS AT ROYAL PRINCE ALFRED HOSPITAL.

AN extra seminar will be given at 1.15 p.m. in the Scot Skirving Lecture Theatre at Royal Prince Alfred Hospital on Friday, December 3, 1954, on the subject of "The Pathogenesis of Megaloblastic Anæmias". The speaker will be Dr. G. M. Watson, from the Department of Pathology, University of Oxford.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1953, as duly qualified medical practitioners: England, John Richard Fountayne, M.B., B.S., 1940 (Univ. Melbourne); Ross, Anne Isabel Brighton, M.R.C.S. (England), 1938, L.R.C.P. (London), 1938.

The following additional qualifications have been registered: Ellis, Frank Fairfax (M.B., B.S., 1937, Univ. Sydney), M.S., 1944, D.L.O. (Univ. Sydney), 1950, F.R.A.C.S., 1953; Greenwell, John (M.B., B.S., 1946, Univ. Sydney), M.R.C.O.G., 1954.

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Feain, Leo Arthur, M.B., B.S., 1954 (Univ. Sydney); Le Lievre, Leslie Ronald, M.B., B.S., 1954 (Univ. Sydney); Linklater, Elizabeth, M.B., B.S., 1954 (Univ. Sydney); McDonald, Anthony John, M.B., B.S., 1954 (Univ. Sydney); McDonald, Keith, M.B., B.S., 1954 (Univ. Sydney); Shortland, Gordon Campbell, M.B., B.S., 1954 (Univ. Sydney); Ward, James Francis, M.B., B.S., 1954 (Univ. Sydney); Woolridge, John David, M.B., B.S., 1954 (Univ. Sydney); Alexander, Kevin Samuel, M.B., B.S., 1953 (Univ. Sydney); Carpenter, John Bolton, M.B., B.S., 1953 (Univ. Sydney); Dunn, Robert Harper, M.B., B.S., 1952 (Univ. Sydney); Hissink, Louis Anne Gerhard, registered in accordance with the provisions of Section 17 (1) (*d*) of the *Medical Practitioners Act*, 1938-1953.

Deaths.

THE following death has been announced:

ROBERTSON.—Allen William David Robertson, on November 8, 1954, at Melbourne.

Diary for the Month.

DEC. 1.—Western Australian Branch, B.M.A.: Council Meeting.
DEC. 1.—Victorian Branch, B.M.A.: Branch Meeting.
DEC. 1.—Victorian Branch, B.M.A.: Branch Council Meeting.
DEC. 2.—New South Wales Branch, B.M.A.: Clinical Meeting.
DEC. 6.—Victorian Branch, B.M.A.: Executive of Branch Council.
DEC. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 8.—Victorian Branch, B.M.A.: Branch Council Meeting.
DEC. 9.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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